# REPORT

OF THE

# HYDERABAD CHLOROFORM COMMISSION,

## WITH A PREFACE

BY

SIR ASMAN JAH, K.C.I.E.,

PRIME MINISTER OF HYDERABAD.

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# PREFACE.

In presenting the Report of the Hyderabad Chloroform Commission to the various Universities and Colleges, and to the Medical profession, I desire to express my regret at the delay which has unavoidably occurred in its production and publication.

This delay has been partly due to the dispersion of the different members who formed the Commission, and partly, also, to the entirely unexpected character of the discussion on the results arrived at by the Commission. Moreover, it was the wish of Surgeon-Major Lawrie that the experimental data of the Commission should be subjected to the test of prolonged clinical experience. This test has been rigorously applied, with results which are set forth in detail in Part IX. of the Report, and which appear to be eminently satisfactory and conclusive.

I agree in the view that free criticism and discussion are essential to the final acceptance of the principles confirmed or brought forward by the Commission. In accordance with this view, everything of importance, which has been advanced during the controversy to which the Commission's recommendations gave rise, has been incorporated in the Report.

At the special request of His Highness the Nizam's Government, the Government of India deputed Surgeon-Major Bomford, of the Indian Medical Service, to serve on the Commission.

I desire publicly to tender my warmest personal thanks, as also those of the Nizam's Government, to the President and Members of the Commission, as well as to the President, Surgeon Patrick Hehir, and members of the first Commission, for the very efficient manner in which they performed their arduous but interesting work. To Dr. Lawrie is due the credit of originating the Commissions, and it affords me great pleasure to convey to him my own cordial acknowledgments, as well as those of His Highness' Government, for his disinterested labours. Our best thanks are also due to Dr. Lauder Brunton and Surgeon-Major Bomford for the thorough and exhaustive manner in which they carried out the wishes of His Highness the Nizam, that the question of the safety of Chloroform should be brought to the proof in Hyderabad. I also desire to record my high appreciation of the work done by Dr. Rustomji on the Commission.

In conclusion, it only remains for me to express the earnest hope that the results arrived at by the Commission may prove of service to the Medical profession, and may be attended with benefit to humanity.

ASMAN JAH.

HYDERABAD DECCAN,
*11th September 1891.*

# REPORT OF THE SECOND HYDERABAD CHLOROFORM COMMISSION.

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## Part I.—THE ORIGIN OF THE COMMISSION.

In presenting the report of the second Hyderabad Chloroform Commission to His Highness the Nizam, the Commission desires to express through His Excellency the Prime Minister, Sir Asman Jah, its grateful sense of the extraordinary and unprecedented liberality and public spirit displayed by His Highness's Government in carrying out a work which is not only of scientific interest, but of the greatest practical importance to the whole human race.

2. The Commission was greatly encouraged in this work by the personal interest shown in it by His Highness the Nizam, who, accompanied by his Staff, visited the laboratory on two special and memorable occasions.

3. The Nawab Munir-ul-Mulk, son of the late Sir Salar Jung, the Maharajah the Peshkar, and the Nawab Fakhr-ul-Mulk also paid visits to the laboratory and witnessed several of the experiments.

4. The same interest which was displayed in originating the Commission was maintained throughout by the principal officers of His Highness the Nizam's Government, especially the Nawabs Mohsin-ul-Mulk, Vikar-ul-Mulk, Imad-ul-Mulk His Highness's Private Secretary, Fatteh Nawaz Jung, and Mr. Furdonji Jamshedji, Private Secretary to Sir Asman Jah.

5. Two Commissions to examine into the alleged dangers of chloroform have been appointed by His Highness the Nizam's Government. The first Commission, which was appointed in 1888, consisted of Surgeon Hehir, I.M.D., President, and two Members, Messrs. J. A. Kelly, L.R.C.P. & S. (Ed.), and A. Chamarette, L.M.S. This Commission was applied for by Surgeon-Major E. Lawrie, Residency Surgeon, Hyderabad, because, having always believed in the truth of Syme's teaching that chloroform can be used judiciously so as to do good without the risk of evil, he desired to show by experiments upon dogs that in death from chloroform the respiration always stops before the heart. This point having been proved, the second Commission was applied for, because it was felt that Syme's principles, which both experience and experiment had shown to be practically sound, must be founded upon a firm physiological basis.

6. The following letter explains the action of His Highness's Government with regard to the first Commission and the lines on which the experiments they performed were conducted :—

From Surgeon-Major E. LAWRIE, M.B., Residency Surgeon, Hyderabad, to Surgeon P. HEHIR, M.D., dated Hyderabad, 18th January 1888.

In accordance with instructions from His Highness's Government, conveyed in Major Gough's letter of the 15th instant, a copy of which is forwarded herewith, I have the honour to inform you that a Committee is appointed, consisting of yourself as President and Messrs. A. Chamarette, L.M.S.; and J. Kelly, as members, to carry out a series of experiments to test the effects of poisonous doses of chloroform on dogs.

These experiments should have an important bearing on the way in which the administration of the anæsthetic ought to be conducted in the human subject.

I would suggest that the experiments of the Commission be conducted on the following lines :—

I. The attention of the Commission should be specially devoted to the effects of chloroform on the circulation and respiration.

II. The chloroform should be administered generally in the same way as it is ordinarily given to patients in the Afzul Gunj and Residency Hospitals.

III. The dose and rapidity of administration should be varied in every possible way, and the admixture of air with the chloroform should also be varied.

IV. At least 100 full-grown dogs should be killed with chloroform, and the points to be specially noted should be—

- (*a*) The time taken to bring the dog fully under the influence of the anæsthetic.
- (*b*) The interval between this and the stoppage of respiration and cessation of the pulse and heart's action.
- (*c*) Whether the heart is directly affected by chloroform, and whether it ever ceases to beat either in slow or rapid poisoning before the respiration stops.
- (*d*) The effects of artificial respiration commenced directly the respiration stops, and at varying intervals afterwards.

V. The details of procedure will be left to the Commission, and you are requested to submit a report of the work of the Commission at any time convenient to yourself before the termination of the official year.

7. The report of the work of the first Commission, which is republished as Appendix A, was embodied in the annual report of His Highness's Medical Department for 1888. The experiments of the Commission led them to conclude " that chloroform may be given to dogs by inhalation with perfect safety, and

without any fear of accidental death, if only the respiration, and nothing but the respiration, is carefully attended to throughout."

8. In forwarding the report to Government, Surgeon-Major Lawrie stated that "the results of the experiments harmonise with the principles necessary for the safe administration of chloroform taught by Mr. Syme, who never had a fatal case, and with my own experience, which is founded upon those principles. I have killed scores of dogs with chloroform, and in every instance death has resulted from failure of the respiratory function. I have also given chloroform in surgery, without a death, for more than twenty years—during the last fifteen years several (five to ten) times every day—and I have never seen syncope or failure of the heart's action produced by it. Finally, it may be mentioned that the Hyderabad Commission have searched the records of accidental deaths from chloroform in Great Britain since the year 1855, and they find that there is not a single death from chloroform recorded, in which it was proved that the respiration alone was attended to throughout the inhalation".

9. The report was forwarded to "*The Lancet*" and other medical journals in due course, and the reception it met with, as well as the circumstances which led to the appointment of the second Chloroform Commission, are rendered clear by the following quotations from "*The Lancet*":

["*The Lancet*," *March* 2, 1889.]

In a report of the recent prize distribution at the Hyderabad Medical School, which appeared in our issue of February 23rd, some remarks of Surgeon-Major Lawrie, M.B., M.R.C.S., of the Bengal Army Medical Service, are mentioned, which deserve some comment. We learn that a Commission had been appointed to investigate the action of chloroform, and that the result of the researches made upon pariah dogs was that these animals were killed from respiratory failure, and in no case did cardiac syncope occur directly. Unfortunately Mr. Lawrie contents himself with bare statements of results, adding that these results tally with his own experience, which he believes to be uniquely large. Mr. Lawrie, as a disciple of Simpson and Syme, arrives at conclusions consonant with the teaching of those great clinicians, but utterly at variance with the experience alike of experiment and practice as carried out in Europe. We should require more than the scanty statements of experiments performed upon dogs—notoriously nonsusceptible to chloroform syncope—before we could accept the conclusions of the Hyderabad Commission when they appear to go in the very teeth of those at which the Commission appointed by the Royal Medical and Chirurgical Society and by the British Medical Association arrived, and, further, are opposed to the careful and painstaking experiments of such scientific observers as Snow, Claude Bernard,* McKendrick, and others too numerous to mention. All those who are familiar with chloroform are well aware that syncope, when primary, as a rule supervenes in the initial stages of inhalation, while secondary syncope due to respiratory embarrassment is the result of accumulation of chloroform in the blood leading to paralysis of the medullary centres, and occurs in a late stage of the administration. The primary syncope is rarely, if ever, possible to induce in dogs, although, unfortunately, it is this form of chloroform heart failure which does occur in human beings, and which it is almost impossible to remedy. While welcoming the attention paid to the subject by the Hyderabad Commission, we cannot but feel that, should the Commission inculcate a disregard of the heart as a factor in chloroform dangers, it will do harm and provoke a slipshod carelessness in the use of that valuable anæsthetic which must in the long run do damage to the cause the Commission has espoused.

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* This is a mistake on the part of the writer in "*The Lancet*," *vide* page 19 of this Report.

["*The Lancet*," *May* 11, 1889.]

## *BY SURGEON-MAJOR E. LAWRIE, RESIDENCY SURGEON, HYDERABAD, AND PRINCIPAL, HYDERABAD MEDICAL SCHOOL.*

In "*The Lancet*" of March 2nd, 1889, page 438, there is an annotation criticising certain remarks of mine on the subject of chloroform in which the writer states that* "all those who are familiar with chloroform are well aware that syncope, when primary, as a rule supervenes in the initial stages of inhalation, while secondary syncope due to respiratory embarrassment is the result of accumulation of chloroform in the blood leading to paralysis of the medullary centres, and occurs in a late stage of the administration," and that unfortunately it is the primary form of chloroform heart failure which occurs in human beings, and which it is almost impossible to remedy. I have no wish to say anything to give offence to those who hold the same views as the writer of the annotation, but I hold that those views are wrong, and that there is no such thing as chloroform syncope.

It is conceivable that syncope may occur in the initial stages of inhalation of chloroform, but in the course of a very large experience I have never met with a single instance of such an accident, and if it ever does occur it cannot be due to chloroform poisoning, though it might be caused by fright or shock. Owing to the numerous accidents that have happened with chloroform, to the discussions prevalent in the profession, and to the mistaken notion that the risk of heart failure is inseparable from its use, the public dread its administration much more than they dread surgical operations, and fainting from mere fright in the early stages of inhalation is no less intelligible than it is easy to prevent, in cases where it is likely to occur, by a preliminary dose of alcohol. On the other hand, it is equally intelligible that syncope may be induced if an operation be commenced in the initial stages of chloroform administration, before the patient is rendered insensible to shock by being brought fully under its influence. With regard to secondary syncope, "*The Lancet*" states that it is due to respiratory embarrassment (through the respiratory centre ?)—which is an indirect cause, and, in the same sentence, that it is the result of paralysis of the medullary centres (circulatory ?) from accumulation of chloroform in the blood,—which is a direct cause.† Both these statements cannot be true. The truth is that secondary syncope has no more real existence than primary syncope. In poisoning by chloroform the heart fails when the respiration ceases, and never before. With the cessation of respiration, the further accumulation of the drug in the blood necessarily ceases, and the heart rapidly or gradually stops beating, as a direct result of the stoppage of respiration, and as an indirect effect of the poisoning with chloroform.

"*The Lancet*" asserts that the statements made in my address are utterly at variance with the experience alike of experiment and practice as carried out in Europe. They are nevertheless based on the principle taught by Syme and Simpson, in Edinburgh, and long before the Hyderabad Commission was formed, I had satisfied myself that they are entirely true.*

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* This sentence should run :—" All those who are familiar with *deaths from* chloroform *in human subjects*"—E. L.

† I may have interpreted the writer's meaning wrongly, but it does not affect the argument.

The Hyderabad Commission was appointed by the Nizam's Government to verify or refute the opinion that if chloroform is properly given it has no injurious or dangerous effect upon the heart. The experiments of the Commission were performed publicly and with every precaution against inaccuracy, and the conclusions they arrived at are irresistible. The chloroform was given in every possible way, and in no case did the heart become dangerously affected until after the breathing had stopped. This is not, as "*The Lancet*" avers, a scanty statement of experiments performed upon dogs: it is a summary of the results of more than two hundred complete experiments carried out by the Commission, and it tallies exactly with my own experience. If direct heart failure were one of the risks of chloroform, it must have occurred in some of the administrations which I have superintended; but I have never seen the heart directly affected by chloroform, nor have I ever seen the inhalation carried so far in human beings as to affect it indirectly. Neither I nor the Hyderabad Commission have any desire to "inculcate a disregard of the heart as a factor in chloroform dangers, and so to provoke a slipshod carelessness in the use of that valuable anæsthetic". Our object is the very reverse of this, as a consideration of the difference between the position we take upon this point and that of those who hold the same views as "*The Lancet*" will show. "*The Lancet*" would trust to the heart and circulation for signals of danger in chloroform administration. Our contention is that, if the administration is ever pushed far enough to cause the heart to show signs of danger, the limits of safety have already been exceeded, and a fatal result must almost inevitably ensue. So far from disregarding the heart as a factor in chloroform dangers, we say that any affection of the heart, either direct or indirect, is the one danger to avoid. But we say further that the respiration invariably gives warnings when a dangerous point is approached, and consequently that it is possible to avert all risk to the heart by devoting the entire attention to the respiration during chloroform administration.

I am well aware that these opinions appear to go in the very teeth of the conclusions at which the Commission appointed by the Royal Medical and Chirurgical Society and by the British Medical Association arrived and "are opposed to the careful and painstaking experiments of such scientific observers as Snow, Claude Bernard, and McKendrick,"† as well as to the teaching of the great London Schools. But this opposition is more apparent than real. "*The Lancet*" states authoritatively that chloroform syncope is notoriously difficult to produce in dogs. The Hyderabad Commission confirms this statement, and says it is impossible to produce it at all. This difference may be fully accounted for by the fact that the experiments of the Hyderabad Commission were numerous, while those quoted by "*The Lancet*" were not only not numerous, but many of them were performed under conditions which impair their value. Moreover there are certain points connected with the effects of chloroform on dogs, which a careful perusal of all their reports has convinced me neither the Commissions nor the distinguished physiologists appreciated. In any case I would ask whether anything can be more discreditable or unsatisfactory to the whole profession than the present position of chloroform. During the last forty years, while enormous progress has been made in every branch of medicine and surgery, the position of chloroform has decidedly deteriorated, and the principles necessary for its safe administration have been lost sight of. From the date of its first introduction there was a divergence of opinion between the Edinburgh and London Schools with regard to the way we should be guided as to its effects. Up to the time of his death Syme taught,* "we are guided as to the effect of chloroform not by the circulation, but entirely by the respiration." At the same time Erichsen was teaching, and still teaches, that† "when fully anæsthetised the patient requires the most careful watching by the person who administers the chloroform; his finger should never be off the pulse, nor his eyes taken away from the countenance of the patient." These are the opinions of two of the most eminent Surgeons that have ever lived, and they are absolutely irreconcilable. Erichsen's opinion has undoubtedly been fortified by the misleading conclusions of the Chloroform Commissions, and of the scientific observers above alluded to, but the Hyderabad Commission has proved that Syme was right; and I have no doubt whatever that, if his principles were acted on universally, all surgeons would find, as he did, that chloroform may be used judiciously so as to do good without exposing patients to the risk of evil. Chloroform administration constitutes, in my humble opinion, the most pressing and important question in the whole range of practical surgery; and if "*The Lancet*" will not accept the conclusions of the Hyderabad Commission, it is incumbent on it to urge the appointment of a European or joint European and American Commission, composed of men of wide experience in chloroform, to confirm or disprove them.

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* *Vide* Lecture reprinted from *Indian Medical Gazette* for March 1889, Appendix D.

† *Vide* page 19 of this Report.

["*The Lancet*," *July* 13, 1889.]

We have received a telegram from Surgeon-Major Lawrie to the effect that His Highness the Nizam of Hyderabad offers to place the sum of £1,000 from his private purse at our disposal, that we may send one or two representatives of "*The Lancet*" to Hyderabad to repeat the experiments made by the Hyderabad Chloroform Commission (*vide* "*The Lancet*," Feb. 23rd and March 2nd, 1889) and to make any others which we may suggest. We await further details of the offer which, we are informed, will be sent by mail.

["*The Lancet*," *September* 21, 1889.]

*The Residency, Hyderabad*, 18*th August* 1889.

THE EDITORS, "*The Lancet*".

SIRS,—I am directed by His Highness the Nizam's Government to offer "*The Lancet*", as the leading medical journal, £1,000 to send out a representative to repeat the experiments of the Hyderabad Chloroform Commission and make any others with the Commission, that you may suggest.

2. The Hyderabad Committee performed a series of experiments of a clinical nature on the effects of chloroform on dogs in 1888, and their conclusions were embodied in a report which was submitted to Government early this year.

3. The experiments of the Commission tend to prove that chloroform has no direct action upon the heart, and though it is hardly to be expected that their conclusions will be accepted as final by the whole of the medical profession, their work may be safely said to constitute an interesting addition to what is already known about chloroform.

4. The Nizam's Government has been advised that if the experiments are continued and amplified by the Hyderabad Commission, associated with a trained scientist whose position and

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* *Vide* "*Lancet*," Vol. I., page 55, for 1855. Syme's original lecture from "*The Lancet*" in which this appears is republished in the appendix.

† *Vide* Erichsen's Surgery, Vol. I., page 14.

attainments will ensure the acceptance of his opinions by the profession, the subject might be threshed out thoroughly, and the question whether chloroform does or does not affect the heart directly, and other questions connected with it, might be settled once for all.

5. His Highness's Government therefore desires to offer £1,000, a cheque for which is forwarded herewith, to the Editors of "*The Lancet*," to send a representative to Hyderabad to repeat and continue the experiments of the Hyderabad Chloroform Commission and make with them any others that "*The Lancet*" chooses to suggest.

6. If His Highness's offer is accepted, the gentleman selected should take a return ticket by P. and O. *via* Brindisi for three months, to be extended if necessary, and he will be the guest of the Nizam's Government during his stay in Hyderabad.

7. The Hyderabad Commission undertake to place themselves entirely at his disposal, and will act under his direction. The Commission will provide all instruments and appliances and everything which may be required for the experiments, and will, without bias, do all in their power to assist the representative of "*The Lancet*" in arriving at the truth.

I have, &c.,
ED. LAWRIE, SURGEON-MAJOR,
*Residency Surgeon*

["*The Lancet*," *September* 21, 1889.]

On another page we print Surgeon-Major Lawrie's letter containing details of His Highness the Nizam of Hyderabad's offer to place at the disposal of "*The Lancet*," as the leading medical journal, the sum of £1,000 to send out a representative to repeat the experiments of the Hyderabad Chloroform Commission, and to make any others that we may suggest. This offer we have cheerfully accepted. There are many young men of great ability and thorough scientific training who could have done this work exceedingly well; but in accordance with the Nizam's desire that we should select a man who is not only a trained scientist, but one whose position and attainments will ensure the acceptance of his opinions by the profession, we have requested Dr. Lauder Brunton, F.R.S., to act as our representative, and he has consented to set out for Hyderabad on October 4th, which is the earliest possible opportunity. Dr. Lauder Brunton has not only devoted much time to pharmacological work for more than twenty years, his first contribution on the action of nitrite of amyl having appeared in our columns in 1867; but the fact that his large work on "Pharmacology and Therapeutics," which appears also in an American edition, has been translated into French, and is now being translated into German, Italian, and Spanish, shows that he is regarded as an authority in other countries as well as our own. It may perhaps be considered as a farther advantage that in this work Dr. Lauder Brunton has very decidedly stated that one of the dangers resulting from chloroform is death by stoppage of the heart. "*Audi alteram partem*" is the motto of an important section of "*The Lancet*," and we think that by getting both opinions regarding the effect of chloroform on the heart represented on the Commission, as they will be by Dr. Lauder Brunton and Surgeon-Major Lawrie, we are more likely to obtain a correct conclusion. The question whether chloroform paralyses the heart or not is one of the greatest possible practical importance, for upon its correct solution the lives of thousands of people and the happiness of thousands of families may depend. Both in Europe and America clinical experience and physiological experiments have led to the conclusion that it has a paralysing action on the heart, while ether exerts such an action in a very minor degree, if at all. In consequence of this ether is now largely used in this country as well as in America for producing anæsthesia in surgical operations, in spite of the greater pleasantness and convenience of chloroform. It is almost impossible to believe that the conclusion at which European and American surgeons and scientists have arrived is, after all, destitute of foundation, and little better than an idle dream. When we find, then, that Dr. Lawrie and the Nizam's Commission have arrived at an entirely opposite conclusion, it is natural that we should hesitate to accept it. A consideration of the report of the Commission appears to show that the number of experiments performed was not only large, but the results were so uniform that the conclusion arrived at, *viz.*, that chloroform does not paralyse the heart, but kills by stopping the respiration, may fairly be taken as correct for the animals experimented on and for the conditions under which the experiments were performed. But if we grant this, we are at once confronted by the next problem: Why do the results of the Nizam's Commission differ from those of European and American investigators? Is it because the experiments in India were carried on in a warmer climate? Or is it because the animals experimented on were peculiarly resistant to the action of chloroform? These questions can only be answered by further experiments, which can hardly fail to be of practical utility, even if they afford only a partial solution of the problem. For Surgeon-Major Lawrie states in his letter, which appeared in our issue of May 11th, that the results of the experiments carried out by the Commission tallied exactly with his own experience. In the correspondence which occurred on the subject in "*The Lancet*" some writers agreed with Dr. Lawrie, while others supported the opposite view. We may perhaps fairly call these two views those of the Edinburgh and London schools. In the Scotch capital failure of respiration is regarded as the chief or only danger, while in the metropolis failure of the heart is more feared. It is quite possible that the surgeons in both cities are right, and that the habits or mode of living of the people may lead to differences in the resisting power of the cardiac or respiratory apparatus respectively. The proportion of gouty patients is much larger in London than in Edinburgh, and when we consider that the natives of India appear to resemble the Scotch in their comparative immunity from cardiac paralysis by chloroform, it will be advisable for the Commission to ascertain, if possible, what the conditions are which enable the heart either in dogs or men to resist the power of chloroform or which lead to its stoppage during the administration of the drug. It may not be possible to work out completely all the questions which may arise, but if the Hyderabad Commission, with the aid of Dr. Lauder Brunton, can settle definitely the question whether chloroform does or does not affect the heart directly, a most important practical object will have been attained by means of the Nizam's generous offer.

10. The opinions held by the medical profession in Great Britain may be fairly judged from the above quotations from "*The Lancet.*" There is no doubt they were and are largely influenced by the experimental results obtained by various observers, and more especially by the Committee of the Royal Medico-Chirurgical Society and of the British Medical Association or Glasgow Committee. Both these Committees arrived at the conclusion, from a very limited number of experiments, that chloroform lowers the blood-pressure, while ether does not. The Glasgow Committee went further, and not only stated that

chloroform lowers the blood-pressure and paralyses the heart, which ether never does, but drew the important conclusion from experiments on one dog only that this action is sometimes exerted in an unexpected and capricious manner.

11. The state of opinion regarding chloroform in America may be inferred from the statements made regarding it in the last edition of H. C. Wood's "Therapeutics," one of the best American works on the subject. He says: "As an anæsthetic chloroform possesses the advantages of quickness and pleasantness of operation, smallness of dose, and cheapness. These advantages are, however, so out-balanced by the dangers which attend its use that its employment under ordinary circumstances is unjustifiable. It kills without warning so suddenly that no forethought or skill can guard against the fatal result. It kills alike the robust and the weak, the well and the diseased; even the previous safe passage through one or more inhalations is no guarantee against its lethal action. Statistics seem to indicate a mortality of about one in three thousand inhalations, and hundreds of utterly unnecessary deaths have been produced by the extraordinary persistence in its use of a portion of the profession. It ought never to be employed except under special circumstances, as when a speedy action is desired in puerperal eclampsia or when the more bulky anæsthetics cannot be transported as in the field in war time."

12. These passages from the work of one of the greatest authorities on the subject in America show that in that continent chloroform is regarded with somewhat of the same disfavour with which it is looked upon in England, and probably upon the same grounds. Although the experiments of the Hyderabad Commission are much more numerous than those made in any previous investigation, yet their number is infinitesimal in comparison with the number of times anæsthetics have been given all over the world, and each administration may be regarded as an experiment on the action of the particular anæsthetic employed. When it is considered that, after so many administrations, or experiments as these may be termed, on the human subject, opinions are so divided in regard to the action of chloroform as at present, we cannot think that the conclusions at which the Hyderabad Commission have arrived will necessarily carry conviction to those who hold opinions opposed to them. The objection may be made that the Hyderabad experiments have been made on the lower animals, and, however correct the Commission's conclusions may be in regard to them, they do not hold for man. To this objection it is a sufficient answer that the fear of chloroform paralysing the heart is based on the results of laboratory experiments, rather than on clinical experience. Deaths have occurred during the administration of

ether as well as of chloroform, and it is not the deaths during operations, but the observations on blood-pressure and on the action of chloroform on the exposed or excised heart which have led to the unfounded dread in the profession that it may paralyse this organ when given as an anæsthetic.

13. But there is still another difficulty ;—however fair and impartial the Hyderabad Commission may try to be, and even though those who differ from them may credit them with care, honesty, and impartiality, its conclusions will always be open to the objection that the Commission may be mistaken, and any mistake on its part would be all the more fatal and disastrous, because its conclusions would be very difficult to disprove since they are founded on such a mass of experimental evidence as has never been collected before, and is hardly likely to be collected again at least for many years. To avoid such a possibility the Commission have tried, as far as possible, to give the experimental data from which their conclusions are derived, so that at any future time and in any part of the world these data will be as available to others as to themselves, and may be used by them either for the purpose of criticising the Commission's conclusions or drawing conclusions of their own. The utility of such a course has been impressed upon the Commission during the progress of this investigation by the example of the Glasgow Committee which they have followed. The Glasgow Committee drew from one experiment alone the sweeping conclusion that chloroform has sometimes an unexpected and capricious effect on the heart's action, and that "the occurrence of the sudden and unlooked-for effects seems to be a source of serious danger."* This conclusion is obviously of the utmost importance in regard to chloroform and, if correct, would justify the condemnation of its use by the American author already quoted. If the Glasgow Committee had only given their conclusions, it would have been very difficult indeed to disprove them; but fortunately they have published a copy of the manometer tracing on which these conclusions are founded, and an inspection of this leads inevitably to the belief that the sudden and unlooked-for effects on the heart's action which the Committee attributed to chloroform are not really due to it at all, but are due to irritation of the vagi quite apart from the action of the anæsthetic, and are the results of accidental asphyxia. The Commission have followed the excellent example of the Glasgow Committee and reproduced by photography the tracings on which their opinion is based, so that all those who look at them and compare them with the tracings of the Glasgow Committee, which they have also reproduced, may judge for themselves and form their own opinions on the subject

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* *Vide* page 115, Experiment No. 148, Observation Q.

14. Dr. Lauder Brunton and Surgeon-Major Bomford arrived in Hyderabad on the 21st of October 1889, and the second Commission was constituted at once as follows :—

Surgeon-Major E. Lawrie, M.B., Edinburgh, *President.*

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| Dr. T. Lauder Brunton, F.R.S.,<br>Surgeon-Major Gerald Bomford, M.D., London,<br>Dr. Rustomji, H. H. the Nizam's Medical Service, | *Members.* |

*Secretary.*—Dr. Bomford.

15. Associated with the second Commission were the President and Members of the first Commission, *viz.* :—

Surgeon P. Hehir, M.D., *President.*

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| Mr. J. A. Kelly, L.R.C.P. & S. (Ed.), L.F.P. & S. (Glas.)<br>Mr. A. Chamarette, L.M.S. | *Members.* |

The Commission is very much indebted to all the gentlemen mentioned, as well as to Mr. William Mayberry, who gave chloroform ; but is especially so to Dr. Arthur Chamarette, to whose energy and fertility of resource the success of the experiments is mainly due. The Commission also desires to thank Mr. Tripp, the Deputy Principal of the Medical School, and Mr. Carroll, His Highness the Nizam's Medical Storekeeper, for much valuable assistance ungrudgingly afforded.

16. The Commission commenced work on the 23rd October and met daily, except on Sundays and holidays, from 7 A.M. till 5 P.M., until the 18th of December, when the experiments were concluded.

17. The Commission was divided into two Committees : one, which will be referred to throughout the report as the Committee, was composed of Drs. Lauder Brunton, Bomford, Hehir and Chamarette ; the other, which will be referred to as the Sub-Committee, was composed of Dr. Rustomji, Mr. Kelly, and Dr. Gay (who volunteered to help), assisted by students.

18. The Committee first of all performed twenty-seven experiments, numbered from 1 to 28, omitting 25, which was a manometer experiment, to test the

work of the first Commission. From No. 29 the Committee were employed principally with blood-pressure experiments. The ordinary experiments, performed without recording apparatus, were then made over to the Sub-Committee, which continued to work in the same room under the supervision of the Commission.

19. The experiments of the Sub-Committee are numbered continuously with those of the Committee, so as to form a consecutive series and facilitate reference.

## Part II.—GENERAL EXPERIMENTS WITHOUT RECORDING APPARATUS.

20. The experiments of the Sub-Committee, together with the first 28 performed by the Committee, form a total number of 430, and are divided into seven sections. These experiments are recorded in a tabular form at Appendix B. 268 dogs and 31 monkeys were killed outright, and 86 dogs and 39 monkeys were subjected to artificial respiration at varying intervals after the natural respiration had been arrested with chloroform. The animals which were killed had chloroform administered to them in every possible way and under every conceivable condition. A large number of dogs were killed just as they were caught in the bazaars; others at various intervals after having heavy meals of meat or farinaceous food or fat; others fasting; others after the administration of Liebig's extract of meat, coffee, rectified spirits of wine or ammonia. Most of these animals were healthy, but some of them had cardiac disease and in many the heart and other organs were rendered fatty by the previous administration of phosphorus. In a large number of cases morphine, strychnine and atropine, singly and in combination, were given by subcutaneous injection at intervals before the inhalation was begun. Chloroform was given with and without inhalers in the vertical and recumbent positions; in glass and wooden boxes; in large and small doses; by being pumped into the trachea with bellows; and in fact in every way that could suggest itself to the Commission.

21. The results in one respect are uniform. In every case where chloroform was pushed the respiration stopped before the heart. The following table shows the interval of time between the cessation of respiration and of the heart's action in dogs and monkeys in uncomplicated cases, and in cases com-

plicated by asphyxia and by the administration of certain drugs subcutaneously before the chloroform inhalation was commenced :—

*Interval of time between stoppage of respiration and stoppage of heart.*

The heart stopped beating under 1, 2, 3, 4, 5, 6, 7, 8, 9, 10, 11, 12, minutes after the stoppage of the respiration.

| | | 1 | 2 | 3 | 4 | 5 | 6 | 7 | 8 | 9 | 10 | 11 | 12 | Total. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| *In Dogs.* | | | | | | | | | | | | | | |
| In uncomplicated cases ... ... ... ... | ... | 1 | 10 | 45 | 53 | 32 | 16 | 8 | 3 | 2 | 2 | 1 | ... | 171 |
| In cases complicated— | | | | | | | | | | | | | | |
| (*a*) by very slow and prolonged administration | ... | 2 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | 2 |
| (*b*) by asphyxia ... ... ... ... | ... | 4 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | 4 |
| *In Monkeys.* | | | | | | | | | | | | | | |
| In uncomplicated cases ... ... ... ... | ... | ... | 2 | 8 | 4 | 2 | 2 | 4 | 2 | ... | 1 | ... | ... | 26 |
| *In Dogs.* | | | | | | | | | | | | | | |
| Injected with cocaine into peritoneum half an hour before chloroform ... | Gr. ¼ ... | ... | ... | ... | 1 | 1 | ... | ... | ... | ... | ... | ... | ... | 2 |
| | „ ½ ... | ... | ... | ... | ... | ... | 2 | ... | ... | ... | ... | ... | ... | 2 |
| | „ 1 ... | ... | ... | ... | ... | 1 | 1 | ... | ... | ... | ... | ... | ... | 2 |
| | „ 1½ ... | ... | ... | ... | 2 | 1 | ... | ... | ... | ... | ... | ... | ... | 3 |
| | „ 2 ... | ... | ... | ... | 1 | 1 | 1 | ... | ... | ... | ... | ... | ... | 3 |
| Do. strychnine ... .. ... | „ $\frac{1}{20}$ ... | ... | ... | 2 | 1 | ... | ... | ... | ... | ... | ... | ... | ... | 3 |
| | „ $\frac{1}{10}$ ... | ... | ... | ... | 3 | ... | ... | ... | ... | ... | ... | ... | ... | 3 |
| | „ $\frac{3}{20}$ ... | ... | 1 | 1 | 1 | ... | ... | ... | ... | ... | ... | ... | ... | 3 |
| | „ $\frac{1}{5}$ ... | ... | ... | ... | 2 | 1 | ... | ... | ... | ... | ... | ... | ... | 3 |
| „ Morphine ... ... ... | „ ¼ ... | ... | 1 | ... | 2 | 1 | 1 | ... | ... | ... | ... | ... | ... | 5 |
| „ „ ... ... ... | „ ½ ... | ... | 2 | 2 | 1 | ... | ... | ... | ... | ... | ... | ... | ... | 5 |
| „ Atropine ... ... ... | „ $\frac{1}{25}$ ... | ... | ... | ... | 2 | 1 | ... | ... | ... | ... | ... | ... | ... | 3 |
| „ „ ... ... ... | „ $\frac{1}{10}$ ... | ... | 1 | 2 | ... | ... | ... | ... | ... | ... | ... | ... | ... | 3 |
| „ „ ... ... ... | „ $\frac{1}{5}$ ... | ... | ... | 1 | 2 | ... | ... | ... | ... | ... | ... | ... | ... | 3 |
| „ „ ... ... ... | „ $\frac{1}{3}$ ... | 1 | ... | 1 | ... | 1 | ... | ... | ... | ... | ... | ... | ... | 3 |
| „ Morphine Gr. ½ strychnine | „ $\frac{1}{50}$ ... | ... | ... | 1 | ... | 4 | ... | 1 | 1 | ... | ... | ... | ... | 7 |
| „ „ „ „ | „ $\frac{3}{100}$ ... | 1 | ... | ... | 3 | 2 | 2 | ... | ... | ... | ... | ... | ... | 8 |
| „ „ „ ½ atropine | „ $\frac{1}{100}$ ... | ... | ... | ... | ... | 1 | ... | ... | ... | ... | ... | ... | ... | 1 |
| „ „ „ „ | „ $\frac{1}{50}$ ... | ... | ... | ... | ... | ... | 1 | ... | ... | ... | ... | ... | ... | 1 |
| „ „ „ „ | „ $\frac{3}{100}$ ... | ... | ... | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | 1 |
| „ „ „ „ | „ $\frac{1}{25}$ ... | ... | ... | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | 1 |
| „ „ „ „ | „ $\frac{1}{20}$ ... | ... | ... | ... | ... | 1 | ... | ... | ... | ... | ... | ... | ... | 1 |
| „ „ „ „ | „ $\frac{3}{50}$ ... | ... | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | 1 |
| „ „ „ „ | „ $\frac{7}{100}$ ... | ... | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | 1 |
| „ „ „ „ | „ $\frac{8}{100}$ ... | ... | ... | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | 1 |
| „ „ „ „ | „ $\frac{9}{100}$ ... | ... | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | 1 |
| „ „ „ „ | „ $\frac{1}{10}$ ... | ... | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | 1 |
| „ Morphine Atropine Strychnine. | | | | | | | | | | | | | | |
| „ gr. ½ | gr. $\frac{1}{100}$ gr. $\frac{1}{100}$ ... | ... | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | 1 |
| „ „ | $\frac{1}{50}$ $\frac{1}{50}$ ... | ... | ... | ... | ... | ... | 1 | ... | ... | ... | ... | ... | ... | 1 |
| „ „ | $\frac{3}{100}$ $\frac{3}{100}$ ... | ... | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | 1 |
| „ „ | $\frac{1}{25}$ $\frac{1}{25}$ ... | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | ... | 1 |
| „ „ | $\frac{1}{20}$ $\frac{1}{20}$ ... | ... | ... | ... | ... | 1 | ... | ... | ... | ... | ... | ... | ... | 1 |
| „ „ | $\frac{3}{50}$ $\frac{3}{50}$ ... | ... | ... | 1 | ... | ... | ... | ... | ... | ... | ... | ... | ... | 1 |
| „ „ | $\frac{7}{100}$ $\frac{7}{100}$ ... | ... | ... | ... | ... | ... | ... | ... | 1 | ... | ... | ... | ... | 1 |
| „ „ | $\frac{8}{100}$ $\frac{8}{100}$ ... | ... | ... | ... | ... | 1 | ... | ... | ... | ... | ... | ... | ... | 1 |
| „ „ | $\frac{9}{100}$ $\frac{9}{100}$ ... | ... | ... | ... | ... | ... | ... | ... | 1 | ... | ... | ... | ... | 1 |
| „ „ | $\frac{1}{10}$ $\frac{1}{10}$ ... | ... | ... | ... | 2 | 1 | ... | ... | ... | ... | ... | ... | ... | 3 |

22. The movement of the heart was in the first 66 cases of the Sub-Committee tested by auscultation, but afterwards by a needle inserted through the chest-wall into the organ and the thoracic cavity was laid open when doubt existed.

23. In the majority of the uncomplicated cases, which include those fed in different ways before inhalation, the heart ceased to act in from two to six minutes after stoppage of the respiration. In one uncomplicated case the heart's action ceased within one minute after the breathing stopped. The heart ceased within one minute after the respiration stopped in two cases where the inhalation was very slow and prolonged, in four cases complicated by asphyxia, and in one where the subcutaneous injection of morphine gr. $\frac{1}{2}$ and strychnine gr. $\frac{3}{100}$ was administered beforehand. The maximum time the heart continued to beat after the respiration ceased, in the experiments of the Sub-Committee, was eleven minutes in a dog and twelve in a monkey.

24. The effects of chloroform do not appear to be interfered with or much influenced by any of the variations in the method of preparation of the animal for or of the administration of, the anæsthetic. There are four general exceptions to this statement. In very slow and prolonged administration, and in cases complicated with partial asphyxia, as well as in one case where one-third of a grain of atropine was administered before the inhalation, the heart stopped very soon after the respiration ceased ; and in all cases where the inhalation was accompanied by struggling the animals became insensible with unusual rapidity. In these cases also the interval between the cessation of respiration and the time of possible restoration by artificial respiration was shortened. (*Vide* tables in Appendix B.)

25. As regards the restorative effects of artificial respiration the Sub-Committee found it was nearly always successful if commenced within thirty seconds after the respiration ceased, very seldom successful if commenced between thirty and sixty seconds after, and always unsuccessful if not begun till after sixty seconds. In forty-four cases, in which artificial respiration proved successful, it was commenced on an average 28·2 seconds after natural respiration ceased. In thirty-eight unsuccessful cases the average was 31·5 seconds.

26. The Sub-Committee formed the opinion that artificial respiration was less successful in restoring the respiration after it had been stopped by chloroform in cases where a subcutaneous injection of morphia was administered before the inhalation was commenced. There were eighteen cases in which this was tried. In the first six artificial respiration was commenced eight seconds after cessation

of natural respiration. Of those five died. In three cases artificial respiration was begun fifteen seconds after the natural respiration ceased, and of these two recovered and one died. In three cases in which artificial respiration was commenced twenty seconds after the respiration stopped, two out of the three died; and, lastly, in six cases where artificial respiration was not commenced till thirty seconds afterwards five died and one was revived. The average of those cases that were revived was 17·6 seconds and of those that died in this series, 26·4 seconds. This opinion was tested by blood-pressure experiments Nos. 162 and 178, the results of which did not support their conclusion.

## Part III.—EXPERIMENTS WITH RECORDING APPARATUS.

27. The experiments carried out by the Committee with self-recording apparatus consist of Nos. 25 and 29 to No. 185 inclusive. In a certain number of cases the animal died accidentally before it was ready to be attached to the manometer, and in others for various reasons the experiment was not completed; but for the sake of convenience they have all been left in the order in which they occurred, and are described *seriatim* in the explanatory notes. A full description of the kymographs, with the aid of which the blood-pressure experiments were carried on, is given in page ; and all these experiments are illustrated by photographs of the kymographic records. A few of these plates illustrate experiments in which the movements of the heart only were recorded by a simple apparatus, which is also described in Appendix A. In every case in which a tracing was obtained it has been without exception preserved, and is reproduced in this report by photography. In this way the whole of the work of the Committee recorded by the manometer has been rendered available for study at any time by any one who is interested in the subject. The photographs are on a reduced scale, but with the aid of a 2 or 3-inch lens and perusal of the explanatory notes, on the page opposite to them, the reader will have no difficulty in deciphering and understanding the remarks recorded on the tracings. The explanatory notes which were written partly at the time of, and partly immediately after, each experiment, were made from the remarks which were recorded on the manometer during the course of the experiments. It is impossible to arrange the manometer experiments so as to group together those which appear to illustrate particular points, because almost every experiment includes a variety of procedures which suggested themselves at the time as likely to elucidate something in the action of the different anæsthetics employed. An index is printed at the end of the plates, in which the anæsthetics or drugs given to the different animals, the operations performed upon them, the actions of interest taken in them,

and the changes of every kind which occurred in their condition, are referred to the number of the experiments in which they took place.

28. The majority of the experiments were made upon dogs or monkeys, and a few upon horses, goats, cats and rabbits.

29. The experiments of the Committee were designed to show the effect upon the blood-pressure, heart, and respiration of the inhalation of chloroform, ether, and the A. C. E. mixture, administered in various ways and under varying conditions. The objects of the Commission were five in number :—

I. To test the suitability and safety of chloroform as an anæsthetic. The experiments with ether and the A.C.E. mixture were instituted principally for the sake of comparison with chloroform on certain points, and it is not pretended that they afford a complete exposition of the action of those agents on the system.

II. The effect of pushing the above-named anæsthetics, (*a*) to a dangerous degree, and more especially until the respiration ceases, (*b*) until death results.

III. The modifications in the effect of these anæsthetics which result from (*a*) asphyxia in varying degrees and produced by various means (*b*) from the use of drugs such as morphine, atropine, physostigmine and others.

IV. The reality or otherwise of the alleged liability during ordinary chloroform administration to the occurrence of primary or secondary syncope or stoppage of the heart, brought about either by shock or through fatty or weak heart, or by hæmorrhage, or by changes in the position of the body. To investigate these points, in the first place, a large number of operations which are reported to be especially dangerous in reference to shock were performed in every stage of anæsthesia, and numerous experiments were also made to show the effect of direct irritation of the vagus. Secondly, a number of animals were dosed with phosphorus before they were experimented on. This caused weakening of the heart by fatty degeneration of its fibres, but at the same time other complicated changes in the whole of the organs of the body not met with in the condition known as fatty heart in human beings. On the other hand, there are conditions often met with in the fatty heart, such as changes of the coronary vessels, which were not produced by the phosphorus.

V. The effect of the anæsthetics above mentioned upon different animals, more especially upon monkeys, as the nearest approach to human beings.

30. The conclusions to which the Commission has been brought by the study of these experiments are the following :—

(1) Chloroform, when given continuously by any means which ensures its free dilution with air causes a *gradual* fall in the mean blood-pressure,* provided the animal's respiration is not impeded in any way, and it continues to breathe quietly without struggling or involuntary holding of the breath—as almost always happens when the chloroform is sufficiently diluted. As this fall continues the animal first becomes insensible, then the respiration gradually ceases, and, lastly, the heart stops beating. If the chloroform is less diluted the fall is more rapid, but is always gradual, so long as the other conditions are maintained ; and however concentrated the chloroform may be, it never causes sudden death from stoppage of the heart. The greater the degree of dilution the less rapid is the fall, until a degree of dilution is reached, which no longer appreciably lowers the blood-pressure or produces anæsthesia.

(2) If the inhalation is interrupted at any stage, the fall of pressure still continues at a rate which depends altogether on the rapidity of the fall while the chloroform was being inhaled. This after-fall is probably due to absorption of a portion of the residue of chloroform in the air-passages after the stoppage of the inhalation. In this way it often happens, if chloroform is given rather freely, that though the respiration may be going on when the chloroform is discontinued, it afterwards stops.

(3) If the administration of the chloroform is stopped at an early stage the pressure very soon begins to rise again, and gradually becomes normal ; but if the chloroform is pushed further, there comes a time, not easy to define, when the blood-pressure and respiration will no longer be restored spontaneously, although the heart continues to beat after the inhalation is stopped.

(4) If the fall has been very gradual, it may occasionally happen that the respiration stops completely, and still the blood-pressure rises again, the respiration recommencing spontaneously in the course of the rise. In the same way when the inhalation has been discontinued, the respiration may stop during the

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* *Vide* especially 168, 169 and 170 and Experiment No. 186 of March 6th, 1890.

after-fall of the blood-pressure and begin again spontaneously. As a rule, if the respiration has stopped, or even becomes slow and feeble at the time when the inhalation is discontinued and artificial respiration is not resorted to, the fall in blood-pressure will continue until death ensues.

(5) There are two conditions which frequently disturb the gradual fall of the blood-pressure, *viz.*, struggling and holding the breath, and it is only by great care that they can be avoided in animals.

(6) Struggling, independently of any change in the respiratory rhythm, appears generally to raise the blood-pressure.* In one case of a dog much weakened from phosphorus† the pressure fell every time he struggled.

(7) When struggling is accompanied, as it often is, by acceleration of the respiration and pulse, especially if the respiration is deep and gasping, it leads to a more rapid inhalation of chloroform, and consequently to a more rapid fall of blood-pressure and a greater after-fall. In order to keep the chloroform cap or inhaler in its place during the animal's struggles, the administrator is obliged to hold it down more tightly over the nose and mouth, and this materially assists in hastening the rapidity of the inhalation, and consequently of the fall in blood-pressure.

(8) The effect of involuntarily holding the breath, which, as anybody can prove by experiment upon himself, must happen when an inhaler saturated with chloroform is first applied to the face, is much more remarkable, the pressure often falling with great suddenness while the heart's action is markedly slowed.‡ As soon as the animal draws breath again the pressure rises as suddenly as it fell, but the gasping respiration which succeeds then causes very rapid inhalation of chloroform with immediate insensibility and a rapid fall of blood-pressure which quickly becomes dangerous.

(9) The combination of struggling with alternate holding the breath and gasping, which results if chloroform is applied closely to the face without sufficient dilution with air, causes violent fluctuations, and then a speedy fall of the blood-pressure, which very soon leads to a dangerous depression with deep insensibility and early stoppage of the respiration. The after-fall under these circumstances is rapid and prolonged. It is this combination of events which causes struggling animals to go under chloroform so quickly.

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* *Vide* Experiment No. 82.
† Do. No. 161. See also Experiment No. 183.
‡ Do. Nos. 103, 119 Fick 2, 157 Fick 4, and many others. See Index under "Breath-holding".

(10) The effect of holding the breath may occasionally cause a temporary fall of blood-pressure after the chloroform inhalation has been stopped* or even when the animal is quite out of chloroform.† This fall is recovered from directly the animal breathes again.

(11) Slight continuous asphyxia, such as is produced by pressure on the neck by straps, a badly fitting muzzle, or hindrance of the chest movements by the legs being too tightly bound down, gives rise to exaggerated and irregular oscillations of the blood-pressure, and slowing and irregularity of the heart's action. If it leads to, or is accompanied by, deep gasping inspiration, it is apt, like anything else which causes this, to increase the intake of chloroform and bring about a rapid decline of blood-pressure.

(12) Complete, or almost complete, asphyxia, as by forcibly closing the nose and mouth‡ or closing the tracheal tube after tracheotomy,§ has an effect similar to, but more marked than, that produced by holding the breath, and the character of the trace corresponds precisely to that produced by irritation of the peripheral end of the cut vagus. The pressure falls extremely rapidly, sometimes almost to zero, and the heart's action becomes excessively slow or even stops for a few seconds. If the Fick trace of Experiment No. 148 be compared with the photographic reproduction of trace A of the Glasgow Committee, it will be seen that they are identical, and that the slow action of the heart with great fall of pressure, which the Glasgow Committee attributed to some capricious action of chloroform upon the heart, was undoubtedly due to asphyxia.

(13) This effect of asphyxia is the result of stimulation of the vagi. The proof of this is (*a*) that the trace corresponds exactly, as stated above, to that produced by direct irritation of the vagus; (*b*) division of both vagi entirely abolishes it; || and (*c*) the administration of atropine which paralyses the vagus also abolishes it.¶

(14) In trace 158 (Fick 4), which was taken during asphyxia after a full dose of atropine, it will be seen that there is an alternately slow and rapid pulse according to the phase of the respiratory movement; but no continued slowing of the heart as in vagus irritation. But there was still a distinct fall of pressure after the atropine when the breath was held, and it was thought that the slowing of the pulse above noted in this condition might be due to the disturbance of the heart from tension in the pulmonary vessels in the absence of respira-

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* *Vide* Experiment No. 157.

† *Vide* Experiment No. 185.

‡ *Vide* Experiments 148, 150, 151.

§ *Vide* Experiment No. 66.

|| *Vide* Experiment No. 150.

¶ *Vide* No. 158, Fick 4.

tory movement rather than to irritation of the vagi. To test this point Experiment No. 184 was instituted. In this experiment the dog's chest was forcibly inflated with bellows connected by a tube with the trachea, and the effect of this proceeding was to cause a fall of pressure and slowing of the heart exactly the same as in involuntary holding of the breath. The dog was then poisoned with atropine, after which inflation of the chest still caused a fall of pressure, but without slowing of the heart (*vide* Fick Nos. 8 and 9). The fall of pressure must be in some degree independent of vagus irritation, which however usually accompanies it.

(15) It only remains to be considered whether the slow action or temporary stoppage of the heart with great fall of pressure produced by vagus irritation is in itself an element of danger in chloroform administration, and if it is not, wherein the danger actually lies.

(16) The experiments in which deliberate irritation of the vagi was carried on during anæsthesia show unmistakably that irritation of these nerves diminishes rather than enhances the danger of anæsthetics. The effect upon the heart is never continuous, and as the vagus becomes exhausted, or when the irritation is taken off, the blood-pressure rises again, as it does when the same result is produced by asphyxia. The slowing of the heart and circulation which is produced by irritation of the vagus by any cause, such as holding the breath in chloroform administration, retards the absorption and conveyance of chloroform to the nerve centres just as holding the breath, whether voluntary or involuntary, prevents chloroform from entering the lungs ; and of itself slowing or temporary stoppage of the heart in chloroform administration is not dangerous.

(17) To answer the second part of the last question in para. 15 is easy enough, if it is kept in mind that the effect of vagus irritation upon the heart is never continuous ; and in chloroform administration as the pressure rises again after the slowing of the heart and temporary fall of pressure produced by any form of asphyxia, violent respiratory efforts with bounding heart's action lead, as in the case of struggling, to a rapid and dangerous inhalation of chloroform and consequent rapid and dangerous decline in blood-pressure. It is, in fact, the temporary exhaustion of the vagi after stimulation that is to be feared, and not the actual stimulation as long as it is continued.

(18) In accordance with this fact it will be found that in chloroform administration neither holding the breath, even if involuntary, or vagus inhibition can be kept up beyond a certain time ; and if the chloroform is not removed from

the face, one or both of two things may happen : (1) when the animal breathes again it takes deep and gasping inspirations, the lungs become filled with chloroform, and an over-dose is taken in with extreme rapidity ; or (2) when the restraining influence of the vagus is taken off the heart, through the irritation ceasing or the nerve becoming exhausted, the heart bounds on again, and the circulation is accelerated in proportion. The blood then becomes quickly saturated with chloroform, and an over-dose is at once conveyed to the nerve centres. The theory which has hitherto been accepted is that the danger in chloroform administration consists in the slowing or stoppage of the heart by vagus inhibition. This is now shown to be absolutely incorrect. There is no doubt whatever that the controlling influence of the vagus on the heart is a safeguard, and that it is the exhaustion of the nerve which is dangerous.

(19) It can be readily understood how a condition in which the pulse is rapid and bounding with high blood-pressure leads to more rapid absorption of chloroform from the lungs, and a more rapid propulsion of the chloroformed blood to the medulla oblongata, and consequently to a more rapid paralysis of the respiratory and vaso motor centres and precipitous fall in the blood-pressure Such a condition is produced in some cases by ether or by division of both vagi* or by a full dose of atropine.† Not only is the poisoned blood carried more swiftly to the vital centres in these cases, but added to this there is the fact that as the heart is already doing its utmost before the chloroform is given, it is unable to stave off by increased work the fall in pressure that occurs when the vaso motor centre is paralysed. On the other hand, it seems clear from Experiment No. 92 that the direct action of chloroform upon the heart's substance is not the cause of the fall of pressure that occurs when it is inhaled.

(20) In Experiment No. 92 repeated injections of 20m. of chloroform were made into the jugular vein, and its effect was not to paralyse the heart, but to produce anæsthesia and a gradual fall of blood-pressure exactly as if the chloroform had been inhaled. In Experiment No. 72, after a considerable amount of ether had been injected into the jugular vein, and a bounding condition of pulse had been produced, the effect of injecting chloroform into the jugulars was much greater, and the fall of blood-pressure much more rapid and dangerous than in the case when chloroform alone was injected. Granting then the truth of Ringer's conclusions from experiments on the frog's heart (which have not been repeated and confirmed by the Commission) that chloroform has a gradual paralysing effect upon the heart's tissue, we must conclude that such an effect, in the degree

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* *Vide* Nos. 151 and 156.

† *Vide* No. 80.

in which alone it could occur in the practical inhalation of chloroform, would rather be a source of safety than of danger.

(21) The Committee discussed the advisability of cutting the vagi some time previous to experimenting on the blood-pressure with chloroform. The effect of this procedure is to cause continuous rapid action and tendency to exhaustion of the heart, as well as to degeneration of the terminal branches of the nerves in the heart if the animal live sufficiently long.[*] Such experiments might be of some interest theoretically, and also have had a practical bearing upon the condition of the heart in certain cases of chronic alcoholism; but the Committee decided not to perform them as it considered the end to be gained did not justify the pain they would have inflicted.

(22) In Experiment No. 178, the case of a dog that had had morphine, remarkable slowing and even temporary cessation of the heart's action occurred again and again at the same moment as the respiration stopped,[*] but the heart invariably recovered itself, and began again to beat regularly before any steps were taken to restore the animal, and without any respiration occurring. We find in this case that it was possible to restore the animal even after unusually long intervals had been allowed to elapse between the cessation of the natural, and the commencement of artificial, respiration. The failure of the heart, if such it can be called, instead of being a danger to the animal, proved to be a positive safeguard by preventing the absorption of the residual chloroform and its distribution through the system.

(23) The effect of artificial respiration after the natural respiration has ceased, is to cause an alternate rise and fall of small amount in the blood-pressure, the trace thus formed upon the drum being a coarse imitation, altered somewhat by the shaking of the table, of the natural respiratory curve. The difference consists chiefly in the fact that the artificial rise and fall is more abrupt than in natural breathing, and that the rise always coincides with expiration or compression of the chest. After artificial respiration has been continued for a certain time, the blood-pressure begins to rise again, and a little later natural respiration returns.

(24) The effect of artificial respiration in restoring an animal after the respiration had stopped was always marked. In a few exceptional cases, such as No. 159, a phosphorus dog, and No. 142, a horse which had an enormous

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[*] *Vide* No. 178, Fick readings 2 and 3, 8, 13, 18, and 26, and compare No. 49, Fick reading 2 and 8, and No 60, Fick reading 3, which are similar cases, but were not so carefully observed.

over-dose, although the artificial respiration was commenced as soon as possible after the breathing was noticed to have stopped, it was not successful.

(25) Complete stoppage of the respiration always means that an over-dose has been administered, and the over-dose may have been so great as to render restoration impossible. It is impossible to say whether, after chloroform has been pushed and then discontinued, the respiration will be restored spontaneously or not, and it is never in any case certain that artificial respiration will restore the natural respiration and blood-pressure, no matter how soon it is commenced after the respiration stops. A great deal depends upon the amount of the after-fall: in some cases, even after the respiration has been restored, the pressure continues to fall and respiration again ceases, and artificial respiration then fails. We thus find respiration restored by artificial respiration while chloroform is still being absorbed, and this tends to show that artificial respiration does not merely pump the chloroform out of the blood, but exerts considerable influence in exciting the natural respiration.

(26) The time which elapses before artificial succeeds in restoring natural respiration varies very greatly. In one case, No. 116, it was continued for eleven minutes before the first natural gasps commenced. This period is undoubtedly prolonged in some cases by a condition of physiological apnœa which renders it unnecessary for the animal to breathe. Consequently, whenever the pressure rose considerably during artificial respiration it was stopped, and the animal then generally breathed after a few seconds.

(27) The time which may be allowed to pass with impunity before commencing artificial respiration also seems to vary considerably. This point was not particularly attended to in the manometer experiments except in experiments 162 and 178, which were instituted to test the truth of the opinion formed by the Sub-Committee that morphine had some slight action in impairing the efficiency of artificial respiration. In these cases the commencement of artificial respiration was postponed for more than two minutes after respiration ceased, and was successful; but this is certainly far above the average interval that can be allowed with safety. The success of artificial respiration in restoring the blood-pressure is, in some cases, very remarkable. In Experiment No. 40 the heart had apparently ceased beating* and the dog was believed by every one present to be dead and yet recovered with artificial respiration. The success in this instance is due to the fact that concentrated chloroform had been pushed for two minutes, regardless of the breathing, and the stoppage of the heart was due

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* *Vide* especially No. 40, Fick reading 12.

to stimulation of the vagus through asphyxia. The animal was therefore easily restored as he was suffering more from asphyxia than from chloroform poisoning.[*]

(28) It corresponds to those cases, which are so often reported, in which dangerous failure of the heart is said to have occurred some minutes after the administration of chloroform had been discontinued, and which are sometimes restored, and sometimes not, by artificial respiration. There is nothing at all sudden about the failure of the heart in these cases, but the attention of the chloroformist, which has been wandering, is suddenly called to the fact that the patient is apparently dead. When the animal was really dead, it was found in some cases that artificial respiration still maintained a small amount of mean pressure in the manometer. In others the pressure seemed to fall to the zero line between each compression of the chest.

(29) The dangers of too vigorous artificial respiration were illustrated in some of the accidental deaths. In one case the liver was badly ruptured, and in another the pleural cavity was full of blood. In three cases, Nos. 80, 92 and 103, rhythmical movements of the diaphragm were noticed after the heart had ceased beating, and after the chest had been opened. It is remarkable that in two of these cases the splanchnic nerve had been divided. The third was a case in which chloroform had been injected into the jugular, and in this case there was a synchronous movement of the jaw as well. In all, death and stoppage of the heart had occurred gradually, and in No. 103 the heart was still irritable. These movements cannot be called respiration, though the last gasp of a dying animal, that ineffective jerk of the diaphragm, which is such a fatal symptom, is very likely in many cases a movement of the same character. Similar movements, which were continued much longer, occurred in Experiment No. 104 after the thorax was opened, while the heart was still beating. Still more remarkable convulsions of the muscles of the jaws, ears, and fore-feet occurred in Experiment No. 167 in the case of a dog that had been poisoned with nicotine. These movements continued at regular intervals for more than ten minutes after death, and were sufficiently forcible to jerk the handles of a pressure forceps fixed on the end of the tongue off the table at each spasm. In a rabbit in Experiment No. 153 the auricles of the heart continued to beat rhythmically for three hours after it was supposed to be dead from chloroform and its thorax had been laid open. Irritability of the heart after death was noticed in many cases, but seemed to be most marked in cases where ether had been used.

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[*] There was a mistake in para. 27 as it originally stood. In the observation at Fick 12 chloroform was administered for two minutes and not for a few seconds only. The error was caused by confusion in the tracing on account of its having been carried twice round the drum at the end of the experiment.

(30) Chloroform injected in the heart through the jugular vein did not cause clotting of the blood as was the case when ether was injected.

(31) In the course of the experiments of the Committee various drugs were administered in order to ascertain if they had any effect in modifying the action of chloroform. The result showed that none of them had any effect in preventing the typical descent of the blood-pressure that occurs when chloroform is inhaled. Atropine, when given in a dose sufficient to paralyse the vagi, of course prevents the action of those nerves in asphyxia, and by increasing the action of the heart it appears to cause a more rapid descent in the blood-pressure when chloroform is inhaled as has been already explained. Morphine appeared in No. 162 to render the rise in blood-pressure that occurred when the chloroform was discontinued slower and less complete, and to bring about a more or less permanent condition of anæsthesia. It may be noted that the animal used in this experiment was a monkey, and in other experiments with monkeys, when no morphine had been given, it was remarked that the animal, after a few inhalations of chloroform, would often lie quite quiet in a state of semi-insensibility for a long time without further inhalations ; still this condition was much more marked in Experiment No. 162 than in any of the others. No action of this kind was noticed in the dog No. 178, but other experiments (Nos. 90 and 94) showed that pariah dogs are very indifferent to the action of morphine, and it is probable that the dose of morphine in this case was insufficient to bring about the condition noted in the monkey. The peculiar behaviour of the heart in No. 178 was not the result of the previous administration of morphine, for a similar phenomenon had occurred in other cases (49 and 60) in which no morphine had been given. Experiments Nos. 162 and 178 prove conclusively that morphine has no effect in shortening the period that may be allowed to elapse between the cessation of natural respiration and the commencement of artificial respiration.

(32) The other drugs used had no effect upon the action of chloroform except when their own special action became the leading feature in the case, as for instance, during the vomiting from apomorphine (104, Fick 9) or the convulsions produced by nicotine (167).

(33) In order to test the alleged danger from shock during chloroform administration, the Committee performed a very large number of those operations which are reputed to be particularly dangerous in this connection, such as extractions of teeth, evulsion of nails, section of the muscles of the eye, snipping of the skin of the anus, &c. In many cases the operation was performed when the

**animal was merely stupefied by the chloroform and not fully insensible.** In such cases a slight variation in the blood-pressure would sometimes occur, such as one would expect from the irritation of a sensory nerve or from the struggling that ensued, but in no case in any stage of anæsthesia was there anything even suggestive of syncope or failure of the heart's action. In thrusting a needle into the heart, there was often a momentary, but well-marked, fall of blood-pressure; but even this was absent in all other injuries. If chloroform really had any power to increase the tendency to shock in operations, it is impossible to believe that it would not have been manifested to some degree at least in one or other of these numerous experiments. The Commission was, however, not content with this negative result, and determined to ascertain the effect of direct irritation of the vagi during continued chloroform administration. The result of such experiments (Nos. 65, 117, and others) proved that inhibition of the heart's action prevented, rather than assisted, the fatal effects of prolonged chloroform inhalation. An animal that was put into a condition of extreme danger (from which it could only be restored by means of artificial respiration) by inhalation of chloroform for one minute, recovered spontaneously and readily after five minutes of chloroform inhalation together with inhibition of the heart by electrical irritation of the vagus carried on simultaneously. In one of these experiments, No. 117, chloroform was pushed for seven minutes; and during continued irritation of the vagus the animals repeatedly came round without artificial respiration. The danger really begins when the irritation is discontinued or fails to inhibit the heart, and thus enables the chloroform in the lungs to be rapidly absorbed and thrown into the system. This danger is certainly increased by deliberately pumping the chloroform into the lungs, by means of artificial respiration, for animals in which this was done, although they showed a tendency to recover when the chloroform and irritation of the vagus were discontinued, afterwards died rapidly.

(34) On another occasion, during Experiment No. 117, the animal was very nearly killed by a comparatively short inhalation of chloroform, owing to the electrodes becoming accidentally short-circuited and failing to keep up the irritation of the vagus. Something similar occurred in No. 177, the effect of the irritation of the vagus passing off, while the chloroform was still being pushed, and thus putting the animal into a condition of extreme and unexpected jeopardy. Nothing could be more striking than these near approaches to accidental death from failure to irritate the vagus efficiently.

(35) Other experiments were made to test the truth of the statement that chloroform increases the action of electrical stimuli applied to the vagus, and

showed conclusively that it has no such effect. In one instance only* the inhibition seemed to be intensified as the chloroform was commenced and diminished when it was discontinued ; but apart from the fact that the supposed effect ceased much too suddenly, a repetition of the experiment on the same and other animals showed that there was in reality no such effect. The increased inhibition in this instance was due to the chloroformist compelling the attendant who was holding the electrodes to change his position and thus making him unconsciously apply them more efficiently. When the chloroformist withdrew they were restored to their former position. This affords an instance of the care that has to be taken in making experiments if one is not to be deceived.

(36) To test the effect of shock due to vaso motor change rather than affection of the heart Goltz's experiment on the frog was repeated on three dogs. In one there was slight lowering of pressure which was not extensive, and in the others no effect was produced at all. Other operations which seemed likely to produce shock, such as violent blows upon the testicle, were singularly devoid of effect. Failing to lower the blood-pressure by any of these methods recourse was had to section of the splanchnics, but the low condition of blood-pressure this produced appeared, like stoppage of the heart from vagus irritation, to be a source of safety rather than of danger during chloroform administration. In this connection Experiment No. 111 may be studied. There was not much external hæmorrhage, but the splanchnics were divided,—a proceeding which, as is often said, bleeds the animal into his own vessels. The pressure was after this extremely low, but chloroform was repeatedly given, and various other actions taken, and then chloroform had to be pushed on a saturated sponge enclosed in a cap for eleven minutes before respiration ceased.

(37) The experiments on dogs that had been dosed with phosphorus for a few days previously show that the fatty and consequently feeble condition of the heart and other organs so produced has no effect in modifying the action of chloroform. The ease with which vagus irritation and the Glasgow trace could be produced in these animals, by even slight degrees of asphyxia (*vide* 148), was very remarkable, but this was equally the case in dogs that had been given phosphorus only a few hours before the experiment and whose organs were not yet fatty (*vide* 156). Many of these cases were in the last stage of phosphorus poisoning, and several of their companions died without any experiment having been performed on them before or on the same day as they did (*vide* the low

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* *Vide* No. 117 Fick reading 6.

state of blood-pressure in No. 163). Numerous attempts were made in these animals to produce shock by operations in the recumbent and vertical positions, but without any more result than in those that were healthy.

(38) The truth about the fatty heart appears to be that chloroform *per se* in no way endangers such a heart, but, on the contrary, by lowering the blood-pressure, lessens the work that the heart has to perform, which is a positive advantage. But the mere inhalation of chloroform is only a part of the process of the administration in practice. A patient with an extremely fatty heart may die from the mere exertion of getting upon the operating table, just as he may die in mounting the steps in front of his own hall door, or from fright at the mere idea of having chloroform or of undergoing an operation, or during his involuntary struggles. Such patients must inevitably die occasionally during chloroform administration, and would do so even were attar-of-roses or any other harmless vapour substituted for chloroform.

(39) The effect of hæmorrhage was tested by opening the femoral artery and allowing a considerable quantity of blood (8 to 12 oz.) to escape. An immediate lowering of the blood-pressure results, and this is very slowly recovered from. Such an accident, however dangerous it may be in itself, in no way affects the action of chloroform, except in so far that a patient who has been nearly bled to death would require less chloroform in his system to put him into a state of anæsthesia. The low condition of his blood-pressure produced by the hæmorrhage would tend to prevent the too rapid intake of chloroform, exactly as in the case of cutting the splanchnics. (*Vide supra* sub. para. 36.)

(40) When the hind feet are lowered on to the floor, so as to place the animal in the vertical position, a considerable fall of blood-pressure in the carotid artery occurs; but when the animal is replaced on the table in the recumbent position the pressure is fully restored. Various operations were performed on animals in the vertical position, but in no case was anything resembling dangerous shock produced. Inversion of the body, so that the animal stands on its head, has exactly the opposite effect,—the pressure rising in the carotid artery and again falling to its former state, when the animal is replaced in the horizontal position. Inversion of the body failed to restore an animal that was in the last stage of chloroform poisoning,* though it raised the pressure in the usual way as long as it was continued. The change in the pressure of

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* *Vide* 106.

the blood of the carotid, which occurs when the position of the body is changed appears therefore to be due simply to the effect of gravity.

(41) As regards the effect of chloroform upon different animals, it may be said to be the same as far as its anæsthetic action is concerned. There are certain peculiarities in its effect on the respiration and circulation connected with its local irritant action on the nostrils and fauces which are interesting to notice. Thus, when concentrated chloroform vapour is applied to the nostrils of rabbits they hold their breath and the heart's action is slowed at once. This is always said to be due to reflex inhibition of the heart from irritation of the nasal branches of the trigeminus reflected through the vagus, and is by no means peculiar to chloroform, but is produced equally by any irritant vapour, such as ammonia or acetic acid.

(42) In some dogs, and especially in those to which phosphorus had been given, stoppage of the respiration and slowing of the heart occurred immediately after the application of the chloroform to the face, or on forcibly pulling out the tongue,[*] and this suggests that the mechanism of cardiac arrest in them is precisely the same as it is in the rabbit. On the other hand, in rabbits, as in all other animals, it is possible to give chloroform so gently that no spasm of the chest occurs, no reflex effect is produced, and then the pressure falls in the same regular curve and with the same succession of phenomena (anæsthesia, cessation of the respiration, and, lastly, cessation of the heart-beat) that was above described as typical of chloroform inhalation.[†]

(43) Goats have a great tendency to hold their breath while inhaling chloroform, and monkeys resemble dogs rather than rabbits, as when ammonia was held before a monkey's (No. 98) nose it did not cause immediate stoppage of the respiration and heart as it does in rabbits.

(44) The experiments with ether show that it is impossible to produce efficient anæsthesia with this agent, unless some form of inhaler is used which thoroughly excludes the air. If an ordinary cap containing a sponge saturated with ether is applied very closely to the face, the animal generally holds its breath and struggles, and we at once get the fall of blood-pressure and slowing of the heart that invariably occurs under these circumstances.[‡] If the ether is continued in this way after the animal has recommenced breathing, a condition of semi-anæsthesia results, in which the cornea is sometimes sensitive and sometimes in-

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[*] *Vide* 185.

[†] *Vide* end of No. 172.

[‡] *Vide* 148 and 261.

sensitive, and the pressure rises and falls alternately to a slight amount and forms a wavy trace, which may be continued right round the drum[*] without any particular change. As soon as air is rigidly excluded the pressure commences to fall gradually exactly in the same way as with chloroform, and with the same succession of phenomena, *viz.*, first anæsthesia, then cessation of the respiration, then of the heart movements, and, finally, death.[*] How far this is due to ether and how far to the results of asphyxia it is impossible to say, but an exactly similar succession of events can be brought about by making the animal inhale carbonic acid gas alone (*vide* 160).

(45) If surgeons choose to be content with a condition of semi-anæsthesia, it can no doubt be produced with perfect safety, though with discomfort to the patient, by ether held rather closely over the mouth. Such a condition of imperfect anæsthesia would never be accepted by any surgeon accustomed to operate under chloroform. If more perfect anæsthesia is required, it can be procured by excluding the air more rigidly; but then there is exactly the same danger as in giving chloroform. How very suddenly and rapidly the pressure may fall and death ensue is well shown by Experiment No. 33. Ether injected into the jugular vein produces a fall of blood-pressure and anæsthesia in the same way as chloroform does (*vide* 93); but in all cases in which it was so injected, large clots were found in the heart immediately after death. It is interesting to note that Claude Bernard seems to have formed a very similar opinion with regard to ether as the following quotations from his work entitled "Leçons sur les Anesthésiques et sur L'Asphyxie," published in 1875 show. The first quotation (page 50) is as follows:—"Aussi un certain nombre de chirurgiens proposèrent-ils d'abandonner le chloroforme pour revenir á l'éther, dont l'usage paraissait moins à craindre. Aujourd'hui encore, les chirurgiens de Lyons emploient préférablement l'éther. On croyait le chloroforme plus dangereux que l'éther parce qu'il était plus actif; mais, en réalité, la fréquence relative des accidents par le chloroforme tenait peut-étre tout simplement à ce que c'était cet agent anesthésique qu'on employait dans l'immense majorité des cas. Plusieurs discussions ont été provoquées par les partisans de l'éther, surtout par les représentants de l'ecole de Lyons, et il a été constaté que l'éther, lui aussi, avait produit un certain nombre d'accidents mortels. Les deux agents anesthésiques usités peuvent donc, l'un comme l'autre, entrainer quelques risques de mort, et la chirurgie humaine à conservé presque partout le chloroforme, dont l'action est plus rapide et plus compléte." The second

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[*] *Vide* 146.

quotation, to be found on page 101 of the same work, runs :—" Quant à l'éther et au chloroforme, leur action est à peu près la même au point de vue physiologique, sauf une différence d'intensité en faveur due chloroforme, ce qui nous fera généralement employer ce dernier corps de préférence à l'éther."

(46) The A. C. E. mixture given gently with plenty of air and the other conditions mentioned before under chloroform produces the typical chloroform trace.* Given freely to a struggling animal, it can produce a very rapid and dangerous fall of blood-pressure.† In Experiment No. 52, Fick 4 shows very perfectly the effect on the heart of holding the breath.

## Part IV.—ACCIDENTAL DEATHS.

31. The notes of the cases of accidental deaths that occurred during our experiments have been left amongst the other notes in the position in which each occurrence took place, and they can be readily found by a reference to the index. The fatal result was brought about either by neglecting to watch the condition of the respiration during or after the administration of chloroform, especially while the carotid artery was being exposed, or from a reckless administration of chloroform in the endeavour to check or prevent struggles. In all the cases of accidental death the usual chloroformist was absent, and no one was attending to the chloroform. The notes would have been more complete if some one could have watched the condition of the animal and noted the gradual but unheeded cessation of respiration without calling attention to it. As it is, one has to be content with the remark that the breathing was noticed to have stopped at some particular time, but there is nothing to throw any light upon the condition during the important period that immediately preceded this discovery. A similar hiatus appears in the account of accidental deaths in the human subject, and is unavoidable. These cases are probably identical with the instances referred to by Snow, " in which animals died in a sudden and what was thought unaccountable manner whilst chloroform was given to prevent the pain and struggles which would be occasioned by physiological experiments."‡ The death was not really sudden, but only rapid, and the result of reckless administration of concentrated vapour in the first instance, and careless neglect of the condition of the respiration in the second. There is no evidence whatever that a single one of them was due

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* *Vide* Experiment No. 45.

† *Vide* Nos. 47 and 52.

‡ *Vide* Snow on Anæsthetics, page 123.

to paralysis or sudden stoppage of the heart, as Snow assumes to have been the case.

32. It must be remembered, in studying the tracings, that, except when it is expressly stated to the contrary, chloroform was throughout administered very freely.* The degree and rapidity of the fall of blood-pressure is in almost all cases much greater than should be the case in administering chloroform to human beings. To avoid complicating the notes the inhaler was kept on much more persistently, with none of those little interruptions while the cornea is being examined, &c., which always occur in practice. The whole series, with few exceptions, may be characterized as examples of reckless administration of chloroform, and accidental deaths would have been much more numerous had it not been that, when once the animal was connected with the manometer, it was kept under the most careful observation. Experiment No. 79 affords a most interesting exception. The chloroformist, though present in body was absent in mind, and failed to observe and report the cessation of the respiration. The chloroform was in consequence pushed much further than it should have been, and the animal died sooner than was intended.

33. These cases are of themselves quite sufficient to show that animals are just as liable to death from the careless administration of chloroform as human beings; and the accidental deaths which occurred during the experiments of the Commission afford the best possible proof that the effects of chloroform are identical in the lower animals and in the human subject. The statement so frequently made that dogs are more resistant to chloroform than human beings is entirely incorrect.

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* It may be noted that one hundred and nine pints of chloroform and eleven pints of ether were used during the experiments of the Commission.

## Part V.—DESCRIPTION OF MANOMETER EXPERIMENTS.

In these experiments the left carotid artery was connected with two manometers by means of a **Y** tube—the Ludwig or slow manometer, the tracing of which runs from left to right, and the Fick or rapid manometer, the tracing of which runs from right to left. The connection of the artery with the Ludwig manometer was continuous, except at short intervals, shown in the Ludwig tracing by a horizontal line with a figure above it. In these short intervals the Ludwig connection was shut off, and a tracing was taken on the Fick. For example, in Experiment 148 at 3h. 32m. 0s. the Ludwig was turned off; this is shown by a horizontal line with the figure 4 above it. The figure 4 in Fick tracing I. shows the tracing taken on the Fick, during the interval marked 4 in the Ludwig at 3h. 32m. 0s.; and so on. As a rule, the Ludwig manometer was cut off by turning the tap while taking a reading with the Fick manometer; but as the drum was allowed to run, each Fick reading is represented by a straight line on the Ludwig tracing.

### October 25th.—No. 25.

Full-sized strong pariah. Into the box with chloroform freely on blotting paper in order to quiet him at 2h. 25m. 10s. Taken out of the box at 2h. 36m. and fastened upon the dog-board, chloroform being given from time to time on Junker's inhaler to keep him quiet. Connection between carotid artery and manometer effected at 2h. 57m. Five tracings with the Ludwig or mercurial manometer* made showing the effects on the blood-pressure of the following proceedings noted :—

(*a*) Ordinary chloroform inhalation.
(*b*) Pushing chloroform until respiration ceased.
(*c*) Artificial respiration until natural breathing was restored.
(*d*) Evulsion of three toe-nails.
(*e*) Ligature of mucous membrane of the anus.
(*f*) Snipping off two portions of anal skin.
(*g*) Free opening of the abdomen and complete ligature of intestine.
(*h*) Continued administration of chloroform until death.

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* The Fick elastic spring manometer was not connected in this experiment.

Respiration ceased at 4h. 30m. 50s., after which the pulse continued feebly, but owing to clotting has not been recorded on the tracing. Death after a few minutes, but not exactly recorded.

### October 26th.—No. 29.

Large cream-coloured dog chloroformed in the box at 2h. 15m. Taken out of the box and put on the dog-board at 2h. 39m. 40s., and kept under chloroform in the ordinary way with Junker's inhaler. Canula inserted into carotid artery at 2h. 51m., and connection made with both the Ludwig and Fick manometers through a Y tube* at 2h. 58m. Trace No. I. Ludwig shows the effect of evulsion of the toe-nails. Trace No. II. Ludwig the effect of snipping the anus, the administration of large doses of chloroform, and of killing the dog with chloroform. A clot formed in the canula at 3h. 15m. 55s. shortly before the dog died. Four readings were taken with the Fick manometer—1 and 2 while the dog was quietly under chloroform, and 3 and 4 during the operation of snipping the anus.

### October 26th.—No. 30.

Full-sized healthy pariah. Into chloroform box at 3h. 39m. Taken out of the box and put on the table at 3h. 4m. 50s. and kept under chloroform in the usual way. Dog fully under at 3h. 50m. 40s., and tying of the carotid artery commenced. Canula inserted at 3h. 58m. 50s., and connection made with the manometers at 4h. 3m.

Tracing I. Ludwig shows effect of a large dose of chloroform ; but it was soon turned off, and the observations until the death of the animal recorded on the Fick tracing I.

### October 28th.—No. 31.

Temperature of the room 18·5 to 20·5 Cent. Full-sized pariah which had a daily dose of phosphorus (about a quarter of a pot of James' phosphorus paste) for the previous three days, and was, in consequence, very weak and thin. Chloroformed in a box at 7h. 35m. Fell down at 7h. 41m. 50s. Placed on dog-board at 7h. 45m. and kept quietly under chloroform. Carotid tied and canula inserted at 7h. 55m. 30s. Connection with manometers made at 8h. 1m., at which time the cornea was still sensitive, and the animal breathing quietly, but very slowly, about 8 times per minute. Two tracings Ludwig, one Fick. Trace I. Ludwig shows effect of (*a*) evulsion of the nails, (*b*) snipping of the anus, (*c*) extraction of teeth, and includes four Fick readings. Trace II. Ludwig (commencing at 8h. 32m. 34s.) shows effect of (*d*) change to the vertical position, and while in that position (*e*) extraction of teeth, and (*f*) administration of chloroform until death. Post-mortem—*Heart* flabby, but otherwise normal ; *lungs*—lower and middle lobes, right side, consolidated ; *liver*—drier than in other animals, mottled, buff-colour; *kidneys*—congested, pyramidal portion of a yellowish colour ; *stomach*—distended with dark fluid, not apparently inflamed.

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* For a full description of the arrangement of the manometers, *vide* Appendix.

*Observations.*

H. M. S.
A. 8 3 50.—Evulsion of nail.
B. 8 8 20.—Anus snipped.
C. 8 15 0.—Evulsion of nail.
D. 8 23 25.—Extraction of tooth.
E. 8 33 0.—Change to vertical position.
F. 8 38 0.—Tooth extracted.
G. 8 41 50.—Artery cut, pressure fell to zero.

In Experiment 31 all attempts to produce shock under chloroform in any stage of the administration failed.

### October 28th.—No. 32.

Large black dog, well nourished and believed to be healthy. Into chloroform box at 10h. 46m. 15s. Struggling violently at 10h. 52m. 30s. Breathing stopped and artificial respiration resorted to until 10h. 54m., when he began again to breathe spontaneously and was placed on the dog-board. More chloroform in cap at 11h. 1m. 30s. Dog struggling. Chloroform stopped at 11h. 2m. 30s. Stertorous breathing. Carotid ligatured at 11h. 7m. 30s. Canula inserted 11h. 10m. 35s. Trace No. I. commenced at 11h. 12m. 20s. Dog struggling. Drum changed and trace No. II. commenced at 11h. 37m. 15s. Canula taken out at 11h. 52m. 15s. Dog not quite dead, heart pulsating, chloroform stopped, and an attempt made to reintroduce canula, but dog dead. Two Ludwig and one Fick tracing. Trace I. Ludwig shows effect of (*a*) administration of chloroform, (*b*) artificial respiration, (*c*) evulsion of nails, (*d*) placing the animal in the vertical position and includes Fick readings Nos. 1 to 6, Trace II. Ludwig shows effect of (*e*) continued administration of chloroform with at the same time (*f*) hæmorrhage from the femoral artery, and includes Fick readings Nos. 7 to 9.

*Observations.*

H. M. S.
A. 11 12 20.—Chloroform pushed to stoppage of respiration and recovery with artificial respiration.
B. 11 18 0.—Chloroform again; and evulsion of nails at 11h. 21m. 0s., 11h. 21m. 40s., 11h. 22m. 0s. and 11h. 22m. 30s. without any effect.
C. 11 23 40.—Chloroform in vertical position.
D. 11 25 0 and 11h. 25m. 40s. evulsion of nails.
E. 11 40 0.—Chloroform: during this administration the femoral artery was opened and the effects of bleeding observed. The experiment ended at 11h. 52m. 15s.

October 28th.—No. 33.

Strong, full-sized dog. Put under chloroform at 2h. 48m. 30s. Dog removed from the box and put on the table at 2h. 56m., and kept quiet with chloroform on the cap from time to time. Carotid tied at 3h. 1m. 30s. Artificial respiration at 3h. 3m. 15s. Breathing naturally again at 3h. 4m. 30s. Canula inserted at 3h. 7m. 30s. Still having chloroform from time to time. Connection made with the manometer at 3h. 8m. 30s. The Ludwig tracing shows the effect of (*a*) ordinary chloroform inhalation, (*b*) pressure on the abdomen, (*c*) inversion of the body, (*d*) administration of ether until death resulted. Post-mortem—Vegetations on pulmonary valves, one of the valves being adherent to the wall of ventricle. The Fick tracing has seven readings in it, which are fully explained on the tracing. Fick 7, taken when ether was pushed, shows exactly the same slowing of the pulse when the respiration has stopped as occurs with chloroform.

Experiment 33 shows the condition of semi-anæsthesia produced by ether held rather closely over the mouth. If more perfect anæsthesia is required it can be procured by more rigid exclusion of air; but then there is exactly the same danger as in giving chloroform. How very rapidly the pressure may fall and death ensue from ether is shown when the anæsthetic was pushed and air excluded at the end of the experiment.

October 28th.—No. 34.

Full-sized, fairly-nourished pariah dog. Chloroform in box 3h. 52m. 15s. Removed from the box and put on the table and kept under chloroform in the usual way. Artery tied at 4h. 8m. 45s. Canula inserted at 4h. 9m. 45s. Connection made with the manometers at 4h. 11m. 30s. Trace I. Ludwig shows effects of ether and chloroform alternately, and finally death from chloroform, and includes 12 Fick readings. Trace I. Fick gives the whole of the 12 Fick readings in four revolutions of the drum.

### October 29th.—No. 35.

Full-sized healthy pariah. Put into the chloroform box at 8h. 18m. 30s. Fallen down at 8h. 22m. 40s. Placed on the dog-board at 8h. 24m. 18s. and kept quiet with chloroform from time to time. Dog was found to be whining even when the cornea was insensitive and breathing stertorous, although chloroform had not been given for some minutes. Carotid ligatured at 8h. 40m. 30s. No. I. Ludwig shows effects of application of bicarbonate of soda to the vagus; electrical stimulation of the vagus and of chloroform administration ; and includes five Fick readings. Tracing No. II. shows the effect of applying an ammonia bottle to the nose and of giving chloroform until death, and includes Fick readings Nos. 6 to 9. Tracing No. III. Ludwig is not preserved, but a partial tracing (III. Fick) reading No. 10 is included in the Fick tracing of dog No. 36.

### October 29th.—No. 36.

Temperature of the room 25 Cent. Full-sized pariah rather thin. Into chloroform box 10h. 45m. 30s. Taken out of box and placed on the dog-board 10h. 56m. 15s. and given chloroform on a cap from time to time. Struggling and crying, although cornea nearly insensitive. Carotid ligatured at 11h. 4m. 5s. Canula inserted at 11h. 10m. 50s., and connection with manometers made at 11h. 26m. 20s. Canula accidentally slipped out of the artery at 11h. 40m. 30s. Dog kept quiet under chloroform and connection again made at 11h. 50m. 30s. Two Ludwig and two Fick tracings during (*a*) application of carbonate of soda to the entire vagus nerve ; (*b*) administration of chloroform ; (*c*) electrical irritation of the right vagus ; (*d*) ligature and division of both vagi ; (*e*) irritation of the peripheral ends of the two vagi separately ; (*f*) irritation of the peripheral end of left vagus when the animal was deeply under chloroform ; (*g*) irritation of the central ends of the two vagi separately ; and, finally, (*h*) administration of chloroform continuously until death.

### October 29th.—No. 37.

Temperature of room 26 Cent. Full-grown common monkey. Given chloroform on a cap while in the cage. Placed on the table 3h. 2m. 30s. and kept quiet under chloroform as before until connection made with the manometer at 3h. 59m. 50s. Two Ludwig and one Fick tracings. Trace I. Ludwig shows effect of (*a*) ordinary chloroform administration, (*b*) evulsion of toe-nail, (*c*) irritation of the entire vagus, and includes five Fick readings. Trace II. shows effects of (*d*) administration of ether, but is much interrupted by accidents to the apparatus. It includes Fick readings 5 to 8. Manometer given up on account of clots at 4h. 53m. Cornea sensitive at 4h. 54m. 15s. Chloroform given at 4h. 54m. 30s. Pulse perceptible but breathing purely laryngeal at 4h. 57m. 45s. No pulse at 4h. 59m. Heart inaudible at 5h., but a thin stick on a needle thrust into the heart continued to vibrate regularly till 5h. 1m.

### October 30th.—No. 38.

Temperature of the room 25 Cent. Full-sized pariah dog. Chloroformed in the box at 7h. 4m. Placed on the table at 7h. 51m. 40s. and kept quiet with chloroform. Jugular vein ligatured. Connection made with manometers at 8h. 45m. 10s. Manometer stopped at 9h. 45m. Dog killed with chloroform. Respiration stopped 9h. 26m. 30s. Pulse stopped 9h. 29m. Heart stopped 9h. 30m. 40s. One Ludwig and one Fick tracing during (*a*) ordinary chloroform administration ; (*b*) irritation of left vagus with the secondary coil distant from the primary 15 centimeters. The Fick tracing consists of three readings :—

1. A failure.
2. Irritation of left vagus, coils 15 cent. distant.
3. Irritation of left vagus, coils 10 cent. apart.

This last reading was, however, interrupted by clotting.

### October 30th.—No. 39.

Large fairly-nourished pariah. Chloroformed and placed upon the table at 10h. 14m. Jugular vein tied and canula inserted into carotid. Connection made with manometer at 10h. 53m. Animal eventually killed with ether. Two Ludwig and two Fick tracings. Trace I. Ludwig shows the effects of (*a*) irritation of the left vagus with the coils 10 cent. apart; (*b*) injection of physostigmine into the jugular vein; (*c*) lifting the vagus from its bed; and includes 6 Fick readings, which show the effect of (*d*) irritation of the entire left vagus; (*e*) application of saturated solution of carbonate of soda to the vagus. Trace II. Ludwig shows effect of (*f*) ligature of left vagus; and (*g*) inhalation of chloroform with rapid but temporary falls of pressure that frequently occur when animals hold their breath at the commencement of the administration. It includes Fick readings 7 to 10, which show the effect of (*h*) pinching the vagus with forceps; (*i*) irritation of the peripheral end of the vagus; (*j*) evulsion of the toe-nails; (*k*) gradual death from ether.

October 30th.—No. 40.

Temperature of the room 26½ Cent. Full-sized pariah dog, the same that had been used in Experiments Nos. 25, 26, and 27, in which experiments he had shown a remarkable power of recovering after chloroform had been pushed until he was to all appearances dead. Chloroformed in box at 1h. 59m. 42s. Lying down at 2h. 5m. and placed on the table at 2h. 5m. 25s., being quite under and with its cornea insensitive. Chloroform given from time to time. Artery ligatured at 2h. 25m. 15s. Artificial respiration at 2h. 36m., but breathing spontaneously again at 2h. 38m. 40s. Connection made with manometer at 2h. 45m. 40s. Artificial respiration was again employed at 2h. 55m. 30s. during an interruption in the tracing, but breathing became natural again at 2h. 56m. Drum changed at 3h. 18m. 30s., and during interruption sciatic nerve exposed. Drum changed again at 3h. 53m. 20s. Three Ludwig tracings and three Fick. Animal just coming out of chloroform.

| | H. | M. | S. | |
|---|---|---|---|---|
| A. | 2 | 45 | 40. | —Inversion of the body, *vide* Fick 3. |
| B. | 2 | 59 | 30. | — Do. do. |
| C. | 3 | 5 | 0. | —Irritation of left vagus (entire) with coils distant 15 cent. It is doubtful if the irritation was efficient. |
| D. | 3 | 7 | 0. | —Irritation of the vagus after administration of physostigmine. |
| E. | 3 | 9 | 30. | —Evulsion of toe-nail. |
| F. | 3 | 29 | 0. | —Irritation of the exposed sciatic nerve with gradually increased strength of current; coil 15, 10, 5, and 0. |

| | H | M. | S. | |
|---|---|---|---|---|
| G. | 4 | 11 | 0. | —Irritation of left vagus, coil 15 after another dose (1-50th of a grain) of physostigmine. |
| H. | 4 | 21 | 0. | —Irritation of sciatic nerve again, coil 15.<br>Irritation of sciatic under increasing coil 15, 10 5 and 0. |
| I. | 4 | 21 | 50. | —Chloroform administered for 35 seconds. |
| J. | 4 | 25 | 0. | —Chloroform pushed. The respiration stopped at 4h. 27m. 15s. After this there were occasional gasps, and at 4h. 29m. 30s. the needle in the heart stopped moving. Artificial respiration was commenced at 4h. 30m. 10s. The animal quickly recovered and breathed naturally at 4h. 31m. 15s. |
| K. | 4 | 37 | 0. | —Ether administered. Respiration again ceased temporarily. |
| L. | 4 | 42 | 0. | —Chloroform pushed again till death. |

Experiment No. 40 was frequently interrupted by clots forming in the tubes. The experiment shows how very remarkable the success of artificial respiration is in some apparently hopeless cases in restoring the circulation. During Fick 12 the heart had ceased beating after the administration for two minutes of concentrated chloroform vapour. Every body thought the dog was dead, but he was easily restored by artificial respiration. Before the observation at Fick reading 12, the animal had been kept under chloroform off and on for two hours and a-half; and for the two minutes immediately preceding the stoppage of the heart it had been pushed regardless of the breathing. The explanation of the easy restoration of the animal by artificial respiration is that he was suffering more from the effect of asphyxia than from the effect of chloroform.

### October 31st.—No. 41.

Temperature of the room 24 Cent. Full-sized healthy pariah with a wound on the buttock. Placed in the chloroform box at 9h. 48m. Fell down 9h. 52m. 30s. Taken out of box and placed on the table at 9h. 56m. Cornea insensible 9h. 57m. Connection made with manometer at 10h. 21m. Three Ludwig and one Fick tracings.

Trace I. Ludwig shows effect of—

| | H. | M. | S. | |
|---|---|---|---|---|
| A. | 10 | 22 | 0. | —Inhalation of chloroform with Junker, squeezed 40 times a minute, at the commencement of which there was a striking but temporary fall of pressure such as often accompanies the struggling and holding breath that ensue when the first whiffs of concentrated vapour are taken into the air-passages. This fall shows that the Junker apparatus interferes with the breathing. |
| B. | 10 | 30 | 10. | —Inhalation of ammonia. |
| C. | 10 | 31 | 30. | —Evulsion of toe-nails. |
| D. | 10 | 33 | 0. | —Extraction of teeth. |
| E. | 10 | 40 | 30. | —Irritation of nostril. |

Trace II. Ludwig shows effects of—

| | H. | M. | S. | |
|---|---|---|---|---|
| F. | 10 | 53 | 5. | —Junker inhalation at the rate of 20 squeezes a minute. |
| G. | 10 | 56 | 0. | —Catching hold and snipping off a piece of the skins of the anus. |
| H. | 10 | 57 | 30. | —Pushing chloroform after respiration has stopped, and then artificial respiration. |
| I. | 11 | 2 | 0. | —Ligature of the right vagus. |
| J. | 11 | 4 | 0. | —Repetition of 3 and 4 and irritation of the central end of the cut vagus after the respiration had stopped. |

Trace III. Ludwig shows effects of—

| | H. | M. | S. | |
|---|---|---|---|---|
| K. | 11 | 20 | 0. | —Junker administration with 10 squeezes per minute. |
| L. | 11 | 22 | 30. | —Irritation of central end of right vagus when dog "well under," but not in danger. |
| M. | 11 | 44 | 0. | —Artificial respiration, delayed for nearly a minute after spontaneous respiration had ceased. |
| N. | 11 | 50 | 0. | —Hæmorrhage to 8 oz. from the femoral artery. |
| O. | 11 | 55 | 0. | —Continued administration of chloroform by Junker at 10 squeezes per minute. |

A clot having been found, the experiment was closed by squeezing the Junker apparatus rapidly 80 times a minute. The heart-beat (indicated by a flag thrust into the heart) continued six minutes after the respiration had entirely ceased and 3m. 15s. after the pulse was imperceptible.

The one Fick tracing includes 7 readings taken at periods indicated on the third Ludwig tracing.

### November 1st.—No. 42.

Temperature of the room 22½ Cent. Full-sized healthy pariah. Chloroformed and placed on the table at 8h. 18m. 20s. Hæmorrhage from the mouth while in the box about 1½ oz., the result of violent strangulation in restraining his movements. Kept quiet with chloroform as before. Struggled violently while taking some chloroform and in consequence was given an overdose. Artificial respiration was resorted to at 8h. 22m. 50s., and was continued till 8h. 34m., but the dog did not recover. Post-mortem—Base of the left lung engorged; both sides of the heart very much distended with venous blood; valves healthy.

This dog's death was undoubtedly the result of the reckless carelessness which had marked the proceedings from the commencement, and was due quite as much to asphyxia as to chloroform.

### November 1st.—No. 43.

Temperature of the room 23½ Cent. Full-sized healthy pariah. Chloroformed in the box as before. Placed on the table at 10h. 5m. and kept quiet, breathing stopped at 10h. 13m. 30s. Artificial respiration until 10h. 14m. 30s. Carotid ligatured at 10h. 17m. 30s. Canula inserted into the carotid at 10h. 20m. Connection made with the manometer at 10h. 25m. Two Ludwig and one Fick tracings.

*Observations.*

H. M. S.

A. 10 25 0.—Administration of chloroform with Junker squeezed 10 times a minute, using the ordinary mouth-piece.

B. 10 30 20.—Administration of chloroform with the lead tube of the Junker thrust into the nostril in place of the mouth-piece, at first at the rate of 10 squeezes per minute, afterwards 20.

H. M. S.
C. 10 35 0.—Squirting a few minims of chloroform into the nostril.
D. 10 44 20.—Administration of ether with a bladder, air being admitted through a side opening every five seconds (the ether administered in this way failed to keep the dog in a state of anæsthesia).

Trace II. Ludwig shows effects of—

H. M. S.
E. 10 52 0.—Junker inhalation, with mouth-piece at 10 squeezes a minute.
F. 11 5 30.—Rapid administration of chloroform with Junker as is done frequently in hospitals, without attending to the signs of danger, while the operation for phymosis was being performed. The dog was dead after 10 minutes, very nearly four drachms of chloroform being used.

November 1st.—No. 44.

Temperature of the room 25½ Cent. Full-sized well-developed pariah dog. Into chloroform at 2h. 1m. Taken down at 2h. 11m. Put on dog board 2h. 11m. 30s. and kept under chloroform as before. Ligature of the carotid at 2h. 17m. 40s. Connection made with manometer at 2h. 26m. 40s. Three Ludwig tracings and two Fick. Trace I. Ludwig shows effects of administration of ether with a bladder, air being admitted through a side opening every ten seconds. (This failed to keep the animal under.) Trace II. Ludwig shows effects of administration of ether with Junker's inhaler provided with a close fitting mouth-piece and also the effect of rapid hæmorrhage to 12 oz., from the femoral artery. Trace III. shows continued ether administration with Junker, and lastly death by pushing chloroform. Fick readings :—

(1) Natural.
(2) During ether administration with bladder.
(3) Do. do. do.
(4) Do. do. do.
(5) Ether with Junker's inhaler ; animal "over".
(6) Ether immediately after 12 oz. hæmorrhage.

(7) Same as 5 ; but a little later the effect of the hæmorrhage still marked.

(8) The same ; only more under the influence of ether.

(9) Just after ether was stopped.

(10) Effect of chloroform pushed.

(11) Do. do.

(12) Do. respiration stopped.

(13) The animal dying.

(14) Dead.

In this experiment (44) the figures (1), (2), (3), &c., refer to the Fick readings.

November 2nd.—No. 45.

Temperature of the room 23 Cent. Full-sized healthy pariah dog. Chloroformed at 9h. 52m. 45s. Fell down at 9h. 58m. 15s. Put on the table at 10h. 1m. 25s. Kept quiet under chloroform. Respiration stopped and artificial respiration at 10h. 15m. Breathing spontaneously at 10h. 15m. 40s. Carotid artery ligatured at 10h. 18m. 5s. Respiration stopped at 10h. 22m. 5s. Artificial respiration until 10h. 23m. 5s. Connection made with manometer at 10h. 29m. 30s. Three Ludwig and three Fick tracings. During trace I. Ludwig ether was administered with bladder inhaler and a closely fitting mouth-piece, air being admitted every fifth and afterwards every tenth respiration, but this failed to keep the dog in a state of anæsthesia. Trace II. shows administration of A. C. E. mixture by a Junker's inhaler, 10 squeezes per minute at first, then 20 squeezes, and finally with rapid squeezes. Trace III. shows the death of the dog, the blood-pressure remaining at 32mm. The three Fick tracings give 15 readings at times marked upon the Ludwig tracings. Total A. C. E. mixture used, 9 drachms.

November 2nd.—No. 46.

Temperature of the room 25 Cent. Full-sized healthy pariah. Chloroformed in box at 2h. 12m. 10s. Placed on the table at 2h. 26m. 30s. and kept quiet with chloroform. Artificial respiration 2h. 23m. 30s. to 2h. 30m. 50s. Artery ligatured 2h. 38m. 55s. Artificial respiration again 2h. 42m. to 2h. 42m. 20s. Connection made with manometers at 2h. 46m. 30s. when the dog was just coming out of chloroform. Two Ludwig and three Fick tracings. Trace I. Ludwig was taken during the administration of A. C. E. mixture with Junker's inhaler, at first at the rate of 10 squeezes a minute, afterwards rapidly. During trace II. Ludwig the A. C. E. administration was continued, but as the effect was not sufficient, the bottle was filled up to 2 oz. with fresh A. C. E., after which the dog died rapidly. The three Fick tracings include 8 readings taken at times noted on the Ludwig tracings. Total A. C. E. mixture used, 13½ drachms.

### November 2nd.—No. 47.

Temperature of room 25½ Cent. Full-sized healthy pariah, to whom ½ a grain of morphine had been given hypodermically about ½ an hour before. Given A. C. E. mixture on a cap 3h. 43m. Cornea insensible 3h. 44m. and placed on the table; quite under. Canula inserted into carotid 3h. 53m. Cornea sensitive and gave more A. C. E. at 3h. 56m. A. C. E. stopped again 3h. 57m. 45s. Connection made with manometer 3h. 57m. One Ludwig and one Fick trace showing the effect of killing the animal with A. C. E. on a cap. The quantity of A. C. E. used was not measured.

### November 4th.—No. 48.

Temperature of room 21 Cent. Unusually large strong pariah dog, which had been fed habitually in the hospital. Chloroformed in the box at 7h. 22m. Put on the table and cornea insensible at 7h. 44m. 10s. Artery ligatured 7h. 56m. 50s. Canula inserted into carotid at 8h. 7m. 15s. Connection made with manometers at 8h. 34m. One Ludwig and one Fick tracings showing the effect of pushing chloroform with the ordinary cap. Recovery appeared for a few seconds to have been effected by artificial respiration, but he again ceased breathing and died. Post-mortem—More fat than usual throughout the whole body. A small cyst in the walls of the æsophagus. Heart healthy.

### November 4th.—No. 49.

Temperature of room 23 Cent. Small-sized healthy pariah dog. Into chloroform box at 9h. 44m. 40s. Placed on the table at 10h. and kept quiet with chloroform. Carotid ligatured at 10h. 9m. Connection with manometers at 10h. 20m. Three Ludwig and four Fick tracings. Trace I. Ludwig shows effect of pushing first chloroform, then ether, and lastly A. C. E., and reviving the dog each time with artificial respiration. Traces II. and III. show a repetition of the above (administration of chloroform, ether, and A. C. E.) and finally death from chloroform. The four Fick tracings include 25 readings at times noted on the Ludwig tracings.

*Observations.*

H. M. S.

A. 10 20 30.—Chloroform on cap.

B. 10 25 0.—Respiration stopped and immediately afterwards the heart's action was arrested. This is what is called secondary syncope. It is not however syncope at all, but is inhibition of the heart due to vagus stimulation, and is a safeguard in that it delays the conveyance of the chloroform to the nerve centres. (*Vide* Fick 2 and compare with Fick 8.)

C. 10 25 10.—Artificial respiration showing the easy restoration of the animal.

D. 10 30 20.—Ether on cap.

E. 10 33 35.—Respiration stopped.

F. 10 33 50.—Artificial respiration and gradual restoration.

G. 10 38 50.—A. C. E. mixture on cap. This observation shows that chloroform, ether, and the A. C. E. Mixture produce precisely similar effects, only varying in intensity.

H. 10 41 30.—Respiration stopped. The same arrest of the heart's action subsequently took place as in observation C. Compare Fick 8 and 9 with Fick 2 and 3.

I. 10 42 0.—Artificial respiration and gradual restoration.

J. 10 49 45.—Observation B repeated. The record is incomplete on account of an interruption from clot.

K. 11 5 0.—Ether, with very little air, on cap. Respiration stopped at 11h. 9m. 30s. Artificial respiration at 11h. 10m. 0s. restored the animal.

L. 11 27 0.—A. C. E. mixture on cap pushed till stoppage of the respiration. Restoration by artificial respiration.

M. 11 39 45.—Chloroform on cap pushed till death.

### November 4th.—No. 50.

Temperature of room 25 Cent. Strong mongrel. Into chloroform box at 2h. 27m. 50s. Placed on the table struggling violently at 2h. 38m., and was given chloroform on a cap. Stopped breathing at 2h. 39m. 50s. Artificial respiration was at once begun and the animal breathed spontaneously at 2h. 42m. Artificial respiration was again necessary almost immediately and failed to restore natural respiration. Post-mortem showed that the abdominal cavity was full of blood from rupture of the liver. The pericardium was adherent; valves healthy. Small abscesses in spleen and liver. This death was the result of excessive zeal and energy in the performance of artificial respiration.

### November 4th.—No. 51.

Temperature of room 25 Cent. Full-sized fairly-nourished pariah. Put into chloroform box at 2h. 50m. 55s. Fell down at 2h. 55m. Placed on the dog-board at 2h. 58m. 15s. Artery ligatured at 3h. 5m. 30s. Connection made with the manometers at 3h. 12m. 40s. One Ludwig and one Fick tracing. The Ludwig tracing shows (*a*) the depressing effect of thrusting a needle into the heart; (*b*) a slighter but distinct depression while struggling at the commencement of ether administration; (*c*) death from ether when pushed with an ordinary cap covered with Mackintosh. The Fick tracing includes seven distinct readings at times noted in the Ludwig trace. The readings along the top of the Fick tracing were produced by a system of Marey's tambours connected with a needle in the heart. Post-mortem—Heart healthy; liver ruptured, but only a small quantity of blood in the abdomen.

| | H. | M. | S. | |
|---|---|---|---|---|
| A. | 3 | 13 | 30. | —Needle thrust into the heart. |
| B. | 3 | 16 | 0. | —Ether administered; the effect of struggling caused slowing of the heart, exactly in the same way as it does under chloroform, and is well shown in Fick I. |
| C. | 3 | 22 | 50. | —Respiration stopped. |
| D. | 3 | 23 | 20. | —Artificial respiration: death. |

### November 4th.—No. 52.

Temperature of room 25½ Cent. Full-sized strong pariah put into the chloroform box at 3h. 38m. Fallen down at 3h. 40m. 50s. Placed on the table at 3h. 42m. 45s. and kept quiet with chloroform. Carotid artery ligatured

3h. 49m. 50s. Canula inserted 3h. 58m. 40s. Connection made with manometers at 4h. One Ludwig and two Fick tracings during—

(*A*) Administration of A. C. E. with marked but intermittent falls in the blood-pressure during struggling.

(*B*) Pushing A. C. E. until respiration stopped.

(*C*) Artificial respiration.

(*D*) Thrusting a needle into the heart.

(*E*) Pushing A. C. E. until death resulted.

*Post-mortem*—Organs healthy. Above two drachms of blood in the pericardium.

*Observations.*

H. M. S.

A. 4 3 0.—A. C. E. mixture: struggling, and consequent irregularities in the fall of blood-pressure of precisely the same kind as those which occur under similar circumstances with chloroform and with ether: a needle was thrust into the heart twice during the observation. The A. C. E. mixture was pushed till stoppage of the respiration at 4h. 5m. 0s. A needle was thrust into the heart again at 4h. 9m. 20s. during revival; and the depressing effect of this operation is well shown in the tracing.

B. 4 11 30.—A. C. E. mixture pushed till respiration stopped: death. In Experiment 52 the effect on the heart of holding the breath is very frequently demonstrated in the Ludwig tracing from 4h. 11m. 35s. to 4h. 12m. 0s. and in the Fick I. tracing 4.

November 5th.—No. 53.

Temperature of the room 23 Cent. Full-sized pariah dog. Into chloroform box at 7h. 47m. 20s. Fallen down at 7h. 57m. Placed on the table at 8h. 2m. 15s. Artery ligatured at 8h. 17m. 20s. Canula inserted at 8h. 23m. 30s. Connection made with manometers at 8h. 27m. 30s. Two Ludwig and two Fick tracings. The tracings show the effect of pushing first chloroform and afterwards ether administered on an impervious cap, and finally killing with chloroform. They also show the effect of thrusting a needle into the heart, and the continued irritation caused by its presence. Post-mortem—

Three drachms of blood in the pericardium. Mitral valves a little thickened. Apex of right ventricle attenuated.

*Observations.*

H. M. S.

A. 8 27 30.—Needle into heart.

B. 8 30 40.—Chloroform pushed till stoppage of the respiration: revival by artificial respiration.

C. 8 38 0.—Needle thrust into the heart.

D. 8 41 0.—Ether administered; struggling and similar irregularities in the circulation, as are observed during struggling under chloroform and the A.C.E. mixture.

E. 9 23 35.—Chloroform pushed: the respiration stopped at 9h. 24m. 30s. The heart continued to beat till 9h. 32m. 20s.

November 5th.—No. 54.

Temperature of the room 24 Cent. Full-sized pariah dog. Chloroformed in box 10h. 9m. 15s. Dog fallen down 10h. 14m. 20s. Placed on the table 10h. 15m. 25s. Stopped breathing 10h. 16m. 10s. Artificial respiration tried without effect. Post-mortem—Organs healthy; two ribs fractured. In this case there was no doubt that chloroform had been carelessly administered while the operation for tying the carotid was being performed.

November 5th.—No. 55.

Temperature of the room 24 Cent. Full-sized healthy pariah dog. Put into the chloroform box at 10h. 29m. 25s. Placed on the table at 10h. 42m. 30s. Artery ligatured at 10h. 51m. 30s. Connection made with the manometers at 10h. 56m. 10s. One Ludwig and one Fick tracing showing administration of ether without air, its failure to keep the dog under and finally death from pushing A. C. E. Post-mortem—two ounces of arterial blood in pericardium. The needle had passed through the lungs into the left auricle. (In this as in previous cases the upper line on the Fick tracing is produced by a system of Marey's tambours connected with the needle in the heart.)

H. M. S.

A. 10 57 0.—Needle thrust into heart. The depressing effect of this measure is shown in Fick reading 1.

B. 11 0 25.—Ether with the air excluded.

C. 11 21 20.—A. C. E. mixture pushed till death.

### November 5th.—No. 56.

Temperature of the room 25½ Cent. Full-sized strong pariah. Chloroform given at 1h. 48m. 30s. Fallen down at 1h. 56m. 30s. Placed on the table at 1h. 58m. 10s. Artery ligatured at 2h. 6m. 45s. Canula inserted at 2h. 11m. 30s. Connection with manometers at 2h. 15m. 15s. In this experiment it was intended to smother the dog with a tin mouth-piece carefully closed, but after death it was found that a sponge with a small amount of chloroform upon it had been accidentally left inside the mouth-piece. One Ludwig and one Fick tracing showing—

*Observations.*

H. M. S.

A. 2 16 20.—Chloroform ; stopped at 2h. 17m. 40s., needle into the heart at 2h. 17m. 50s.

B. 2 20 5.—Simple asphyxia with a very small quantity of chloroform, accidentally left in the inhaler. At 2h. 27m. 0s. the heart's action was arrested, probably owing to vagus stimulation, for 15 seconds (*vide* Fick 7). The heart stopped finally at 2h. 30m. 0s.

Fick reading 8 shows clearly the gradual cessation of movement in the needle thrust into the heart. Post-mortem examination showed general venous congestion ; small hæmorrhage in the lungs ; a small cyst in the wall of the heart, and a large suppurating lymphatic gland in the wall of œsophagus.

### November 5th.—No. 57.

Temperature of room 26 Cent. Full-sized pariah. Chloroformed in box 2h. 37m. 15s. On the table 2h. 42m. 30s. Artery ligatured 2h. 49m. Connection made with manometer 2h. 53m. One Ludwig and two Fick tracings. An attempt was made to simply smother the dog with the padded mouth-piece ; but complete asphyxia could not be produced, and he was eventually killed with A. C. E. mixture pumped into the mouth-piece with Junker's inhaler. Fick readings 4, 6, and 7 show very well the slowing of the heart produced by asphyxia. Post-mortem—Intense venous congestion.

*Observations.*

H. M. S.

A. 2 53 40.—Partial asphyxia.

B. 3 11 5.—A. C. E. mixture pumped into the mouth-piece with Junker's inhaler till death. Respiration ceased at 3h. 16m. 0s. and the heart stopped at 3h. 20m. 15s.

### November 5th.—No. 58.

Temperature of the room 26 Cent. Full-sized powerful pariah dog. Into the chloroform box at 3h. 28m. 15s. Fallen down at 3h. 32m. 10s. Placed on the table at 3h. 33m. 45s. and kept quiet with chloroform. Artery ligatured at 3h. 40m. Canula inserted at 3h. 43m. 30s. Connection made with manometers at 3h. 44m. 35s. One Ludwig and one Fick tracing. Asphyxia by means of the mouth-piece was again attempted, but as this failed to kill the dog owing to leaks, chloroform vapour was pumped into the closed mouth-piece with Junker. Unfortunately directly after this a clot formed in the tube.

### November 6th.—No. 59.

Temperature of room 22·5 Cent. Full-sized pariah. Into chloroform box at 8h. 20s. Fell down at 8h. 9m. 45s. Placed on the table at 8h. 10m. Temperature in rectum 100·4 F. More chloroform at 8h. 17m. 35s. and again at 8h. 23m. 30s. Canula inserted at 8h. 26m. 30s. Connection made with the manometers at 8h. 32m. 20s. One Ludwig and two Fick tracings showing the effect of asphyxiating the animal by holding a carefully-sealed inhaler over his muzzle. The act of thrusting the needle into the heart did not cause any fall of pressure. Temperature in the rectum 99 F. at the end of experiment. Post-mortem presented all the appearance of asphyxia. The needle had entered the heart through the auriculo-ventricular groove and just reached the interior behind one of the semi-lunar valves. No blood in the pericardium.

### November 6th.—No. 60.

Temperature of room 23½ Cent. Large size dog. Into chloroform box at 10h. 3m. Fell down at 10h. 12m. Placed on the table at 10h. 13m. Temperature in the rectum 100·4 F. A little chloroform given at 10h. 21m. Artery ligatured at 10h. 22m. 10s. Canula inserted at 10h. 25m. Connection made with manometer at 10h. 27m. One Ludwig and one Fick tracings. Asphyxia was first produced by smothering the dog with an empty inhaler, and then chloroform was pumped into the still air-tight inhaler. Death resulted very rapidly. From Fick reading 3 it was thought that the heart had stopped simultaneously with the respiration, but, on opening the thorax, the heart was found to be still beating, and it continued to do so for some minutes. Post-mortem appearances those of chloroform poisoning. Temperature after the experiment 99 F.

*Observations.*

A. 10h. 28m. 0s.—Smothering with an empty inhaler. The irregular fall of pressure due to asphyxia, and to asphyxia combined with chloroform, is seen in Fick readings 1 and 2.

B. 10h. 32m. 5s.—Chloroform pumped into the air-tight inhaler. Fick 3 shows the very rapid fall of pressure due to vagus stimulation in an animal already partially asphyxiated, and how quickly chloroform-poisoning and paralysis of the respiratory centre occurs under these circumstances. From Fick 3 and the absence of pulse it was thought by everybody present that the heart had altogether stopped, simultaneously with the respiration, at 10h. 33m. 0s., but when the thorax was opened at 10h. 36m. 30s., three minutes and-a-half after the breathing stopped, the heart was found to be beating vigorously, and it continued to do so till 10h. 39m. 50s., nearly seven minutes after the cessation of respiration.

Experiment No. 60 affords an excellent example of the remarkable slowing and even temporary cessation of the heart's action, which occurred again and again throughout the Commission's enquiry at the same moment as the respiration stopped. The heart always recovered itself and began to beat regularly again before any steps were taken to restore the animal and without any respiration taking place. (*Vide* Experiments Nos. 49, 162 and 178, and compare with 60). The failure of the heart, if such it can be called, instead of being a danger to the animal, is a safe-guard by preventing the absorption of residual chloroform from the lungs and its distribution through the system. If the arrest of the heart in these experiments is due, as it appears to be, to vagus stimulation, there is no doubt that the inhibiting action benefits the organ and, besides resting it, acts on it as a tonic.

November 6th.—No. 61.

Temperature of room 24 Cent. Full-sized pariah dog. Put into the chloroform box at 10h. 42m. Fallen down at 10h. 55m. 30s. Placed on the table at 10h. 57m. 45s. Temperature in the rectum 99·2 F. Artery ligatured at 11h. 4m. A little chloroform given at 11h. 9m. Connection made with the manometers at 11h. 10m. 15s. Temperature after death 98·2 F. Two Ludwig and two Fick tracings showing the effect of giving ether to an asphyxiated animal. The extraordinary fall of pressure, which occurred at the commencement of the ether inhalation, is noticeable. It was the accompaniment probably of struggles and holding breath, though this was not noted on the tracing. As ether did not appear to be going to kill the animal in a reasonable time, chloroform was pumped into the inhaler and death ensued rapidly. The second Ludwig tracing is imperfect, on account of the float not working freely in the tube of the manometer. The needle was found in the apex of the left ventricle.

### November 6th.—No. 62.

Temperature of the room 25·5 Cent. Full-sized strong pariah dog. Put into chloroform box at 2h. 25m. Fallen down at 2h. 32m. 30s. Put on the table at 2h. 34m. 30s. Temperature in the rectum 101 F. Artery ligatured at 2h. 40m. 30s. Canula inserted into the carotid at 2h. 44m. Connection made with the manometers at 2h. 46m. One Ludwig and one Fick tracing. A little chloroform was given at first to quiet the animal and diminish the excursions of the needle. Afterwards the empty inhaler was applied and made as air-tight as possible, and, lastly, A. C. E. was pumped into the inhaler until death ensued. The end of the experiment was interrupted by a clot. Temperature in the rectum 99 F. after the experiment.

### November 6th.—No. 63.

Temperature of the room 25·5 Cent. Medium-sized strong pariah dog. Put into the box and chloroform given at 3h. 19m. 10s. Fallen down at 3h. 30m. 15s. Brought on to the table at 3h. 31m. 15s. Temperature in the rectum 101·6 F. Chloroform from time to time to keep him quiet. Artery ligatured at 3h. 41m. 25s. Canula inserted into carotid at 3h. 42m. 50s. Glass tube tied in to the trachea at 3h. 50m. Connection made with the manometers at 3h. 53m.

One Ludwig and one Fick tracing, showing the effect of opening and closing the tracheal tube (*vide* especially Fick readings 3 and 4). Chloroform was afterwards pumped into the tracheal tube by means of Junker's inhaler until death ensued.

### November 7th.—No. 64.

Temperature of the room 21 Cent. Full-sized pariah dog, rather thin. Put into the chloroform box at 7h. 46m. 30s. Fallen down at 7h. 52m. 30s.

Placed on the table at 7h. 53m. and given a little chloroform from time to time to keep him quiet. Artery ligatured at 8h. 5m. 10s. Temperature in the rectum 98·2 F. A loose ligature placed under both vagi. Connection made with the manometer at 8h. 20m. 15s. Three Ludwig and three Fick tracings. Ludwig I. is during (*a*) a short administration of chloroform ; (*b*) ligature of the right vagus after the chloroform was stopped, causing very marked and continued depression ; (*c*) artificial respiration ; (*d*) division of the right vagus ; (*e*) peripheral irritation of the right vagus, electrically (coils distant 10 cent.), with at the same time the pushing of chloroform on an ordinary cap for a little over two minutes ; (*f*) artificial respiration and gradual recovery. Ludwig tracing II. is during (*g*) peripheral irritation of the right vagus (Fick reading 9) ; (*h*) irritation of right vagus. peripheral as before, and pushing of chloroform at the same time (Fick reading II.) for about one minute ; (*i*) gradual recovery without artificial respiration ; (*j*) the same experiment again, irritation of right vagus and pushing chloroform, for over two minutes ; (*k*) recovery again without artificial respiration, but in the course of recovery, irritation of the right central vagus ; and (*l*) ligature and section of the left vagus. Ludwig tracing III. is during (*m*) irritation of the periphery of both vagi with pushing of chloroform at the same time for about two minutes ; (*n*) opening and placing a tube into the trachea; (*o*) chloroform accidentally squirted into the trachea and gradual death of the animal. The time of the 22 Fick readings is noted on the Ludwig tracings. Temperature in the rectum, after death 98·4 F.

### *Observations.*

A. 8h. 20m. 30s.—Chloroform administered. Irritation of right vagus at 8h. 22m. 0s. causing almost total arrest of the pulse from 8h. 22m. 0s. to 8h. 23m. 45s. During this period there were only 25 pulsations registered, and most of these pulsations occurred during the last 45 seconds (*vide* Fick 2). Chloroform was pushed to stoppage of the respiration and the animal was restored by artificial respiration. The vagus was divided at 8h. 27m. 0s.

B. 8h. 29m. 50s.—Irritation of the peripheral end of the right vagus when the animal was out of chloroform. The slowing of the heart and great fall of blood-pressure caused by stimulation of the vagus are well shown on the Ludwig tracing. The administration of chloroform was commenced during more or less complete inhibition of the heart (*vide* Fick 5 and 6) from 8h. 30m. 10s. to 8h. 32m. 0s. The respiration ceased at 8h. 32m. 0s. and the chloroform cap was removed. Artificial respiration was not begun until 8h. 33m. 55s. and natural breathing was restored at 8h. 39m. 0s.

C. 8h. 53m. 30s.—Irritation of peripheral end of right vagus followed by rapid chloroform administration at 8h. 53m. 40s. (*vide* Fick 11). At 8h. 54m. 15s. the vagus irritation was stopped, and the administration of chloroform was discontinued at 8h. 54m. 30s. At 9h. 1m. 30s., when the animal was coming round spontaneously, irritation of the peripheral end of the right vagus was again commenced while chloroform was pushed simultaneously and continued until 9h. 3m. 40s. The respiration entirely ceased at 9h. 3m. 10s. and recommenced spontaneously at 9h. 4m. 10s. The chloroform cap was removed, but the blood-pressure continued to fall, owing to the residual chloroform in the lungs, until 9h. 4m. 40s. (*vide* Fick 13). The central end of the right vagus was irritated from 9h. 6m. 55s. until 9h. 7m. 50s. and this caused *very slow respiration.* The left vagus was divided at 9h. 9m. 40s.

D. 9h. 14m. 20s.—Electrical irritation of peripheral ends of both vagi, causing an immediate fall of the blood-pressure almost to zero. Chloroform administration was pushed during complete inhibition of the heart's action at 9h. 14m. 30s. There was entire absence of pulse-tracing for more than one and-a-half minutes, the blood-pressure remained nearly at zero, and the breathing became slow. The irritation and the administration of chloroform were both stopped at 9h. 16m. 0s. The blood-pressure rose immediately to nearly its former height. It then gradually fell, exactly as it does in chloroform administration with normal breathing, and rose again spontaneously at the end of one minute. The fall of pressure after the cessation of vagus stimulation constitutes the most interesting phenomenon in this observation. From 9h. 14m. 20s. to 9h. 16m. 0s. cardiac inhibition, with sudden and prolonged fall of blood-pressure, was caused by stimulation of the vagi. The arrest of the circulation, due to stoppage of the heart, prevented the chloroform, which from 9h. 14m. 30s. was saturating the air deep down in the lungs, from getting into the blood. But when the circulation was resumed at 9h. 16m. 0s. the chloroform was forthwith taken up by the blood, and the respiration was no longer a factor in the process, except to eliminate it from the lungs. The effect of the uncontrollable absorption of chloroform into the blood was, not to give rise to any paralysis or weakening of the heart, but simply to produce the ordinary regular and gradual fall of the blood-pressure, which is associated with narcosis of the nerve centres in the medulla, in normal chloroform inhalation. The trachea was opened at 9h. 19m. 0s. and a tube inserted at 9h. 19m. 50s.

E. 9h. 25m. 0s.—Chloroform was accidentally squirted into the trachea. Respiration stopped at 9h. 28m. 45s.; slight thoracic movements occurred at 9h. 31m. 25s ; a needle was inserted into the heart at 9h. 33m. 30s; and the heart-movements were perceptible until 9h. 35m. 20s. The artery was cut and the pressure fell to zero at 9h. 36m. 0s.

### November 7th.—No. 65.

Temperature of the room 23½ Cent. Full-sized pariah, but very old. Put into the chloroform box at 10h. 43m. 30s. The dog escaped from the box, but was put into it again at 10h. 45m. 15s. Fallen down at 10h. 56m. Placed on the table at 10h. 56m. 45s. and given enough chloroform from time to time to keep him quiet. Temperature in the rectum 100·8 F. at 11h. 4m. 20s. Artery ligatured at 11h. 7m. Canula inserted into the artery at 11h. 6m. Loose ligature placed under both vagi. Connection made with manometers at 11h. 20m. 5s. Three Ludwig and three Fick tracings during (*a*) rapid chloroform administration until respiration ceased and for about a quarter of a minute afterwards, *i.e.*, for 1 minute in all ; (*b*) gradual recovery without artificial respiration ; (*c*) double ligature and division of the right vagus ; (*d*) irritation of the peripheral end of the right vagus, together with pushing of chloroform by the ordinary cap for 2½ minutes. The distance of the coils was changed from 10 cents. to 5 cents. in the middle of the experiment ; (*e*) gradual recovery without artificial respiration ; (*f*) irritation of the central end of the right vagus (Fick reading 7) ; (*g*) pushing chloroform for nearly one minute and stopping the respiration, necessitating resort to (*h*) artificial respiration ; (*i*) irritation of the central end of the right vagus again (Fick 10); (*j*) irritation of the periphery of the vagus again with chloroform pushed as before for five minutes ; (*k*) spontaneous tendency to recovery assisted after 3½ minutes by a short artificial respiration ; (*l*) ligature and division of left vagus ; (*m*) pulling at the vagi ; (*n*) irritation of both vagi, pushing chloroform for just over 5 minutes, while artificial respiration was employed whenever the respiration stopped so as to ensure the chloroform entering the lungs. Afterwards the dog took a few shallow respirations and then died in spite of artificial respiration.

Temperature in the rectum just after death 98 F.

### *Observations.*

A. 11h. 22m. 50s.—Pushed chloroform ; breathing stopped at 11h. 23m. 34s.; chloroform cap removed 11h. 23m. 50s ; breathing restored spontaneously without artificial respiration.

B. 11h. 30m. 0s.—Chloroform pushed with simultaneous irritation of peripheral end of right vagus. The slowing of the pulse due to vagus stimulation is shown in Fick 4. Breathing stopped at 11h. 32m. 35s., and recommenced spontaneously at 11h. 33m. 20s. Fick 7 shows the tracing of the respiration and pulse during irritation of the central end of the vagus. The respiration was not stimulated, but arrested by the irritation of this portion of the nerve.

C. 11h. 38m. 5s.—Chloroform pushed till 11h. 38m. 55s., when the respiration stopped and the pulse tracing disappeared (*vide* Fick 8). The animal was restored by artificial respiration. The central end of the right vagus was irritated from 11h. 41m. 50s. to 11h. 42m. 20s. The effect in slowing the respiration, and of this on the pulse, is well shown in Fick 10.

D. 11h. 51m. 55s.—Irritation of the peripheral end of right vagus followed at 11h. 52m. 0s. by chloroform. The irritation and chloroform were kept up till 11h. 57m. 0s. Respiration stopped at 11h. 54m. 0s. There were a few gasps at 11h. 57m. 10s. Artificial respiration was not commenced till 12h. 0m. 40s. The animal recovered. The first respirations are seen in the Ludwig tracing between 12h. 1m. 0s. and 12h. 2m. 0s. The effects of stimulation of the vagus in slowing the heart and circulation are displayed in readings 13, 14, 15, 16 and 17 Fick. The left vagus was divided at 12h. 4m. 55s.

E. 12h. 12m. 45s.—Irritation of the peripheral ends of both vagi and chloroform administration kept up till 12h. 18m. 0s., the chloroform being inhaled by artificial respiration when natural respiration failed. Death ensued. Experiment No. 65 proves that inhibition of the heart's action, due to stimulation of the vagi during continued chloroform administration, prevents rather than assists the fatal effects of prolonged chloroform inhalation, and that lowering of the blood-pressure either suddenly or gradually without weakening of the heart is in no sense a danger.

### November 8th—No. 66.

Temperature of the room 21·5 Cent. Full-sized pariah dog. Into chloroform box at 9h. 25m. 30s. Fallen down at 9h. 33m. Placed on the table at 9h. 35m. 45s., and given chloroform now and again. Canula inserted into left carotid. Loose ligatures placed under both vagi and a large-sized glass tube tied into the trachea. Connection made with the manometers at 10h. 6m. The animal was first asphyxiated and then given ether through the tracheal tube by means of Junker's inhaler. Death ensued rapidly.

### November 8th.—No. 67.

Temperature of the room 23 Cent. Full-sized pariah dog. Into the chloroform box at 10h. 39m. 15s. Fallen down at 10h. 48m. 0s. Placed on the table at 10h. 51m. 30s. Temperature in the rectum 99·0 F. Ligature of the carotid at 11h. 3m. 30s. Tube placed in trachea and loose ligatures under both vagi. Connection made with manometers at 11h. 16m. One Ludwig trace and one Fick (one Fick reading is incorporated with the Fick readings of 68). Chloroform was administered by the Junker inhaler attached to the tracheal tube and death occurred very rapidly. Before the chloroform was given the sciatic nerve was irritated. Artificial respiration was tried for several minutes in vain, though it kept up a certain amount of blood-pressure, and ammonia was held opposite the opening of the trachea. Post-mortem examination showed that there was nothing abnormal in the heart or other organs. Rupture of the liver had occurred and there was much blood in the peritoneum, but this can only have occurred after the death of the dog, as artificial respiration was very gently applied for a long time. Temperature in the rectum after death 99·4 F.

### November 8th.—No. 68.

Monkey (fair-sized Macacus). Chloroformed at 2h. 10m. 45s. in box. Temperature of the room 24·5 Cent. Fallen down at 2h. 23m. 15s. On the table at 2h. 27m. 45s. and kept quiet with chloroform from time to time.

Temperature in rectum 100·6 F. Carotid artery ligatured at 2h. 38m. Canula inserted at 3h. 40m. 30s. Ligature placed under both vagi. Junker's lead tube inserted into an opening in the trachea and the chloroform administered through it. Connection made with manometers at 3h. 5m. 30s., but stopped again almost immediately on account of bleeding from the artery below the canula. Connection again made at 3h. 19m. 15s. One Ludwig and one Fick tracing. During the tracings numerous attempts were made to irritate the right vagus, but it is not probable that the nerve had really been exposed. The canula slipping at 3h. 38m., an attempt was made to place it in the right carotid, but failed, and the experiment was closed by killing the monkey with chloroform.

November 8th.—No. 69.

Temperature of room 25 Cent. Full-sized pariah dog just brought in. Into chloroform box at 4h. 6m. Fallen down at 4h. 12m. 20s. Placed on the table at 4h. 13m. 30s. Artery ligatured at 4h. 18m. 50s. Breathing stopped at 4h. 19m. 40s. Artificial respiration until 4h. 20m. 30s. Temperature in rectum 103·2 F. Connection made with manometers at 4h. 29m. 30s. Two Ludwig and two Fick tracings.

*Observations.*

H. M. S.
A. 4 32 0.—Electrical irritation of the entire left vagus with the coils 5 cent. apart.
B. 4 33 0.—The same, Fick reading 2.
C. 4 35 10.—The same, only coils approached to 0. Fick 3.
D. 4 39 10.—The same.
E. 4 39 55.—Administration of chloroform, during which the right vagus was accidentally irritated in the process of exposing it.
F. 4 41 30.—Electrical irritation, coils 0, of the entire right vagus, the animal being nearly out of chloroform.
G. 4 42 10.—The same. Fick reading 6.
H. 4 50 0.—Exposure of the sciatic nerve.
I. 4 51 15.—Irritation of the right vagus as before with simultaneous irritation of the sciatic nerve.
J. 4 54 0.—The same, Fick reading 7.
K. 4 54 30.—Ordinary administration of chloroform.
L. 5 1 0.—Repetition of I and K when the dog was "well under," *vide* Fick 9.

H. M. S.

M. 5 5 0.—Administration of ether on a sponge placed in a tin inhaler with the air-holes open.

N. 5 9 0.—Repetition of I, K, and M, under ether.

O. 5 13 0.—The same. *Vide* Fick 11 ; the effect of irritating the vagus not marked as before.

P. 5 16 0.—Division of the right vagus.

Q. 5 20 0.—Irritation of the peripheral end of the right vagus while still under ether.

R. 5 21 0.—Ditto when dog "quite out". Chloroform was then pushed until death resulted, the heart stopping at 5h. 25m. But the tracing was interrupted by a clot in the tube.

November 9th.—No. 70.

Temperature of the room 20 Cent. Large-sized pariah dog. Into chloroform box at 9h. 42m. 50s. Fallen down at 9h. 45m. 15s. On the table at 9h. 47m. Left carotid artery ligatured at 9h. 53m. 15s. and canula inserted into it. Loose ligature under right carotid and both vagi. Connection made with manometers at 10h. 2m. 40s. Two Ludwig and one Fick tracings.

*Observations.*

H. M. S.

A. 10 3 30.—Clamping the right carotid artery so that both carotids were obstructed.

B. 10 6 40.—Administration of ether in an open inhaler, through which there was free entrance of air during the clamping of the carotid and after the clamp was taken off.

C. 10 17 35.—Ligature and division of the right vagus.

D. 10 19 10.—Peripheral irritation of the right vagus with a 5 cent. coil. Cornea insensitive.

E. 10 20 15.—Repetition of D, *vide* Fick reading 7.

F. 10 22 0.—Exposing sciatic nerve.

G. 10 24 10.—Irritation of the sciatic nerve, coil 5. No effect.

H. 10 37 35.—Opening the trachea and fixing a tube in it.

I. 10 39 55.—Administration of ether on a blotting paper cone held over the end of the tracheal tube. This, however, failed to keep the dog insensitive, although a little ether was squeezed into the tube.

H. M. S.

J. 10 48 40.—Continued ether administration with the end of the tube closed by compressing the cone and afterwards by squirting a little ether into the trachea.

K. 10 51 50s.—Artificial respiration in vain.

Temperature after death 97·6 F. Post-mortem—Blood on both sides of the heart; large plug of venous blood at the bifurcation of the trachea extending far down into the bronchi, probably the result of blood running into trachea, post-mortem, when the tube was taken out, as this was observed to happen in No. 71 after the tube was taken out. The upper readings on the Fick manometer tracing were produced by a system of Marey's tambours connected with a pin stuck into the diaphragm.

November 9th.—No. 71.

Temperature of room 22·5 Cent. Full-sized pariah. Weight 30 lbs. Into box at 11h. 2m. 40s. and chloroform commenced. Fallen down at 11h. 6m. 10s. On table at 11h. 9m. 45s. Kept quiet with chloroform. Respiration stopped at 11h. 12m. Artificial respiration directly this was observed and continued till 11h. 12m. 50s, when spontaneous respiration recommenced. A good deal of hæmorrhage from vessels in the neck. Two veins and one small artery ligatured. Artery ligatured at 11h. 25m. Canula inserted at 11h. 26m. 30s. into left carotid. Loops under both vagi, the right carotid and trachea. Temperature in rectum 99·4 F. Connection made with manometer at 11h. 33m. 55s.

Two Ludwig and one Fick tracing.

*Observations.*

H. M. S.

A. 11 35 40.—Clamping of the right carotid so as to close both carotid arteries.

B. 11 38 20.—Exposing the sciatic nerve.

C. 11 38 50.—Ordinary chloroform administration.

D. 11 40 40.—Irritation of the sciatic coil 5 when hardly under chloroform, which was being pushed.

E. 11 41 30.—Ligature and division of right vagus.

F. 11 47 10.—Irritation of sciatic again after administration ot chloroform had ceased, but dog still under.

G. 11 47 30.—Irritation of the sciatic again, but in the middle increasing the strength by moving the coil to 0 (Fick reading 6). The irritation of the sciatic produced no effect.

H. 11 50 0.—Opening the trachea and placing a tube in it.

H. M. S.

I. 11 52 0.—Giving chloroform on a cone of blotting paper attached to the tracheal tube.

J. 12 0 40.—Administration of ether in the same way for a few seconds by mistake.

K. 12 4 0.—Pulling at the right vagus.

L. 12 4 50.—Irritation of the peripheral end of the right vagus, coil 5 during continued pushing of chloroform.

M. ..... Irritation of both right vagus and sciatic during continued pushing of chloroform. Stoppage of the respiration at 12h. 8m. 0s. and spontaneous recovery at 12h. 9m. 45s.

N. 12 10 30.—Squirting ½ a drachm of chloroform into the tracheal tube, and—

O. 12 11 30.—Irritation of the right vagus again. The end was obscured by a clot in the tubes.

Temperature at death 98 F.

November 9th.—No. 72.*

Temperature of the room 23·5 Cent. Full-sized pariah dog. Weight 30 lbs. Into the chloroform box and chloroform given at 2h. 21m. 50s. Placed on the table at 2h. 25m. 35s. and kept quiet with chloroform. Stopped breathing at 2h. 24m. 15s. Artificial respiration until 2h. 35m. 45s. Temperature 108·8 F. in the rectum. Left carotid ligatured at 2h. 36m. 45s. Right jugular vein ligatured and canula inserted at 2h. 52m. Connection made with the manometer at 2h. 53m. 30s. One Ludwig and one Fick tracing showing the effect of injecting ether and afterwards chloroform into the jugular vein. Temperature after death 102·2 F. Post-mortem—the blood in the left side of the heart was clotted.

*Observations*—

A. 2h. 58m. 45s.—Fifteen minims of ether injected into the jugular vein: no appreciable effect. At 2h. 59m. 40s. twenty minims of ether were again injected. Between 3h. 1m. 0s. and 3h. 1m. 30s. one drachm of ether was injected into the jugular vein and the blood-pressure fell very rapidly. The respiration stopped at 3h. 2m. 0s. and the Fick reading 2 shows almost total arrest of the pulse tracing exactly similar to that caused

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*** Nos. 72 and 73 may be grouped with Nos. 77, 91, 92 and 93.**

by vagus stimulation after cessation of the respiration from chloroform poisoning. At 3h. 2m. 20s. artificial respiration was commenced and natural respiration was restored at 3h. 4m. 30s.

B. 3h. 8m. 30s.—Twenty minims of chloroform injected into jugular vein. The effect was precisely the same as that of the intravenous injection of ether. Fick reading 4 with chloroform is identical with Fick reading 2 with ether. This and other experiments, when pure chloroform and ether were injected into the large veins of the neck, show that even pure chloroform or ether in the blood do not stop the heart till after they have produced narcosis and paralysed the respiration.

The most important point in Experiment 72 is that after a considerable amount of ether had been injected into the jugular vein and a rapid condition of pulse had been produced, the effect of injecting chloroform into the jugulars was much greater, and the fall of blood-pressure was much more rapid and dangerous than in the case when chloroform alone was injected.

November 9th.—No. 73.*

Temperature of the room 24 Cent. Full-sized pariah. Weight 32 lbs. Put into chloroform box at 3h. 28m. Fallen down at 3h. 35m. 40s. Placed on the table at 3h. 36m. 30s. Temperature 102·2 F. in rectum. Jugular vein ligatured and canula inserted. Left carotid ligatured at 3h. 45m. Connection made with the manometers at 3h. 50m. One Ludwig and two Fick tracings during injection of ether (Fick 2) into jugular vein, afterwards chloroform (Fick 5) and again ether (Fick 9), which brought about death. Temperature after death 100 F. Post-mortem—Small clot in the left ventricle, which was filled with venous blood, clot in superior vena cava. Serous fluid in the pericardium.

*Observations.*

H. M. S.

A. 3 53 10.—Injection of twenty minims of ether into deep jugular vein; repeated at 3h. 53m. 35s., 3h. 54m. 5s., and 3h. 54m. 30s. The effect is shown in Fick reading 2.

B. 3 59 40.—Injection of twenty minims of chloroform into jugular vein; the effect is shown in Fick reading 5 and is identical with that produced by ether.

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* Nos. 72 and 73 may be grouped with Nos. 77, 91, 92 and 93.

H. M. S.

C. 4 5 55.—Injection of twenty minims of ether. See Fick reading 9. The injection was followed by rapid fall of pressure; the respiration stopped at 4h. 6m. 40s.; gasping took place at 4h. 8m. 0s.; the thorax was opened at 4h. 9m. 0s., and the heart was found still, with a clot in the left ventricle. The disastrous effect of injecting ether after chloroform is well shown in this experiment.

November 11th.—No. 74.

Temperature of the room 19·5 Cent. Rather small pariah dog. Weight 21 lbs. Into chloroform box at 8h. 13m. 30s. Fallen down at 8h. 17m. Placed on the table at 8h. 21m. 50s. Temperature in the rectum 100·4 F. Left carotid ligatured at 8h. 28m. 10s. Right jugular vein ligatured at 8h. 33m. and canula inserted into left carotid 8h. 44m. Connection made with the manometers at 8h. 46m. 15s. Two Ludwig and three Fick tracings.

*Observations.*

H. M. S.

A. 8 47 30.—Ordinary chloroform administration.

B. 8 48 20.—Thrusting a needle into the heart.

C. 8 49 25.—Irritation with a 10 cent. coil of the right vagus entire, (Fick reading 3).

D. 8 51 5.—Chloroform again rapidly. Stoppage of respiration and recovery with artificial respiration.

E. 9 0 0.—Injection of atropine 1-50th grain into the jugular vein.

F. 9 6 40.—Chloroform administration again during struggling; the respiration again stopping and necessitating artificial respiration.

G. 9 14 25.—Irritation of the right entire vagus.

H. 9 16 50.—Injection of 1-25th grain of atropine into jugular vein.

I. 9 22 5.—Irritation of right vagus again when animal under influence of atropine. Coils moved from 10 to 5 and then to 0 (Fick 13).

K. 9 34 30.—Pushing chloroform until death.

Heart was still beating 3 minutes after the pulse became imperceptible. Temperature after death 97·4 F.

Post-mortem—Two drachms of blood in the pericardium; no clot in heart.

November 11th.—No. 75.

Monkey (Macacus). Weight 15 lbs. Into chloroform box at 10h. 29m. 50s. Fallen down at 10h. 34m. 30s. Placed on the table at 10h. 47m. and kept quiet with chloroform from time to time. Temperature in the rectum 101·8 F. Loop under both vagi. Artificial respiration 10h. 57m. 15s. to 10h. 58m. 5s. Left carotid ligatured at 11h. 1m. 30s. Canula inserted at 11h. 4m. 50s. Connection made with manometer at 11h. 18m. 10s. Two Ludwig and two Fick tracings.

*Observations.*

| | H. | M. | S. | |
|---|---|---|---|---|
| A. | 11 | 19 | 0. | —Ordinary chloroform administration. |
| B. | 11 | 21 | 20. | —Holding ammonia before the nostril. |
| C. | 11 | 24 | 40. | —Holding acetic acid before the nostril. |
| D. | 11 | 26 | 0. | —Dropping a few minims of chloroform into the nostril. |
| E. | 11 | 45 | 50. | —Irritation of the entire right vagus, coil at 10 cents. (Fick 4). |
| F. | 11 | 48 | 0. | —The same with the Ludwig manometer connected. |
| G. | 11 | 53 | 0. | —Irritation of the entire right vagus, coil at 5 (Fick 5). |
| H. | 11 | 58 | 40. | —Irritation of the right vagus, coil at 0 (Fick 6). |
| I. | 12 | 2 | 20. | —Thrusting needle into the heart. |
| K. | 12 | 3 | 45. | —Pushing chloroform until death. |

Needle entered the left ventricle. Temperature soon after death 97 F.

November 11th.—No. 76.

(*In the presence of His Highness the Nizam.*)

Pariah dog. Weight 37 lbs. Temperature of the room 23·5 Cent. Into chloroform box at 2h. 21m. 36s. Fallen down at 2h. 28m. 30s. Placed on the table at 2h. 30m. 50s. and kept quiet with chloroform. Artificial respiration at 2h. 40m. 25s. and until 2h. 44m. 15s. Carotid ligatured at 2h. 41m. 5s. Temperature in the rectum 100 F. Loop under both vagi. Connection made with manometer at 2h. 48m. 25s.—Two Ludwig and two Fick tracings.

*Observations.*

| | H. | M. | S. | |
|---|---|---|---|---|
| A. | 2 | 48 | 25. | —Ordinary chloroform administration in several instances. |
| B. | 2 | 51 | 50. | —Irritation of right vagus repeatedly (Fick 2—4). |
| C. | 3 | 34 | 5. | —The same after the injection of atropine into the jugular veins (Fick 6). |

H. M. S.

D. 3 40 0.—Thrusting needle into heart.

E. 3 42 30.—Inhalation of ammonia.

F. 3 43 0.—Pushing chloroform until after respiration had completely stopped, and then

G. 3 46 0.—Restoration by artificial respiration.

H. 3 48 20.—Pushing chloroform until death resulted.

Temperature after death 99·4 F.

November 12th.—No. 77.

Temperature of the room 18·5 Cent. Medium-sized pariah 26 lbs. Into chloroform box at 7h. 27m. 12s. Fallen down at 7h. 36m. 50s. On the table at 7h. 38m. 50s. Artery ligatured at 7h. 50m. 10s. Temperature in the rectum 99·2 F. Canula inserted into the left carotid at 7h. 58m. 50s. Right jugular ligatured at 8h. 3m. 10s. Connection made with manometer at 8h. 13m. Three Ludwig and one Fick during repeated injection of ether into the jugular vein in doses of first 20m., then 40m., and 60m., and finally 120m. One injection of 60m. was also made into the thigh subcutaneously. When the pulse was no longer perceptible, a needle was thrust into the chest, but did not reach the heart and consequently did not move. The dog was still gasping occasionally until the thorax was opened at 9h. 12m., when the heart was found to have ceased beating, but rhythmical waves in the heart-wall occurred until 9h. 24m., although the heart had been removed from the body and cut open. Temperature after death 98 F. Post-mortem—Large clot in left ventricle. Right side full of clots.

*Observations.*

H. M. S.

A. 8 13 30.—Ether 20 minims injected into jugular vein ; no effect.

B. 8 18 20.—Repeat injection of 20 minims of ether ; this injection was followed by slight fall of blood-pressure.

C. 8 19 50.—Another 20 minims of ether injected into the jugular vein. The fall of blood-pressure again occurred to a greater extent.

D. 8 24 10.—Repeat ether injection. A still further fall of pressure is noticeable and the recovery from this fall was slow.

E. 8 27 20.—Forty minims of ether injected into the jugular vein. A very rapid and deep fall of blood-pressure took place and the pulse tracing disappeared (*vide* Fick 4). During recovery at 8h. 32m. 0s. and 8h. 35m. 0s. the blood-pressure twice rose and fell again in a remarkable manner without any apparent cause. (Compare with Experiment 117, Observation G at 9h. 23m. 50s. to 9h. 25m. 15s). The condition of respiration was not recorded at these times.

F. 8h. 40m. 0s.—Repeat injection of 40 minims of ether into jugular. During recovery at 8h. 47m. 40s. a drachm of ether was injected subcutaneously into the thigh. Recovery was arrested and a slight fall of pressure took place; but no marked effects were produced.

G. 8h. 50m. 20s.—Injection of 60 minims of ether into jugular vein. This was followed by rapid fall of pressure and complete loss of pulse tracing, and the respiration stopped at 8h. 53m. 0s. The pupils were widely dilated. The animal began gasping at 8h. 54m. 40s. and recovery gradually took place, but the blood-pressure was not fully restored.

H. 9h. 7m. 40s.—Repeat injection of ether into jugular vein. This time 120 minims were injected. The pressure rapidly fell again and the respiration stopped for 20 seconds, after the injection was concluded. A few gasps took place at 9h. 10m. 0s. ; the thorax was opened at 9h. 12m. 0s. and rhythmical wave-like movements were observed in the ventricular walls of the heart, which continued until 9h. 24m. 0s. after the heart had been removed from the body. Large round clots were found in the left ventrical extending into the aorta ; and the right side was full of clot.

Experiment 77 shows that the effect of ether is precisely the same as that of chloroform, only less intense. Like chloroform, ether, when efficiently given, produces first narcosis, then stoppage of the respiration and then death. All the observations in the experiment show that every time ether was injected into the blood, a fall of blood-pressure was produced ; and yet it is the practice now-a-days to inject ether subcutaneously when chloroform poisoning occurs. It would be just as sensible to inject chloroform subcutaneously in cases of poisoning from ether. Cases have even been recorded recently where ether has been injected subcutaneously in *ether poisoning*. This manifestly dangerous and unsound principle of treatment arises from the fact that ether is regarded as a safe stimulant, whereas it merely quickens the heart's beats. Experiment 77 shows that it is not a reliable stimulant under any circumstances.

November 12th.—No. 78.

Monkey (Macacus). Weight 10½ lbs. Temperature of the room 21 Cent. Into chloroform box at 10h. 4m. 30s. On to table at 10h. 9m. 23s. and kept quiet with chloroform. Temperature in rectum 98° F. Artery ligatured at 10h. 34m. 50s. Loops under both vagi. Connection made with manometer at 10h. 46m. 30s. Two Ludwig and one Fick tracing.

H. M. S. *Observations.*

A. 10 49 0.—Ordinary chloroform administration.

B. ...... Violent struggling with great irregularity of blood-pressure.

C. 10 54 20.—Dropping chloroform into nostril ; effect of struggling again shown.

D. 10 55 20.—Pushing chloroform until respiration stopped.

E. 11 6 20.—Mechanical irritation of the right vagus.

F. 11 7 0.—Electrical irritation of the entire right vagus, coils 10 cent. distant (Fick 8).

G. 11 10 50.—Ligature of the right vagus.

H. 16 26 35.—Division of the same.

I. 11 29 40.—Irritation of the peripheral end of the right vagus, coil at 10 (Ludwig tracing).

J. 11 31 0.—The same, only coil at 5 (Fick 9).

K. 11 33 0.—Injection of atropine into the peritoneal cavity.

L. 11 39 40.—Irritation of the peripheral right vagus again, coil 5 (Fick 11).

M. 11 45 30.—The same, only coil 0.

*N.B.*—The effect upon the blood-pressure of pushing chloroform was not nearly so great as in previous experiments, though the animal was very readily put " under ".

Hopeless clotting stopped the manometer at 11h. 56m. 20s., and the monkey was given chloroform finally with the view of killing it. Respiration stopped at 12h. 4m. 40s. ; pulse perceptible until 12h. 6m. 30s. Heart flag-moving until 12h. 9m. 55s. Thorax opened and heart still beating rhythmically at 12h. 11m. 35s., a little blood passing into the aorta at each beat. The contractions became partial and ineffective at 12h. 18m. 40s., but were re-established by compressing the septum and continued until 12h. 28m. Irregular flickering continued even after this for some minutes. Temperature 98 F. in the rectum.

## November 12th.—No. 79.

Good-sized pariah, weight 33 lbs., that has had two doses of phosphorus—one yesterday, the other to-day, each 1-16th of a box of James' beetle paste. Into chloroform box at 2h. 9m. 34s. Fallen down 2h. 23m. 39s. Placed on the table at 2h. 24m. 26s. Respiration stopped at 2h. 24m. 45s. Artificial respiration until 2h. 27m. 39s. Artery ligatured and canula inserted at 2h. 36m. 13s. Temperature in the rectum 100 F. Connection made with the manometer at 2h. 39m. 30s. Two Ludwig and one Fick tracing.

| | H. | M. | S. | *Observations.* | |
|---|---|---|---|---|---|
| A. | 2 | 41 | 50. | —Change to the vertical position of the body. | |
| B. | 2 | 43 | 10. | —Chloroform administration while in the vertical position. | |
| C. | 2 | 44 | 45. | —Ammonia inhalation | do. |
| D. | 2 | 46 | 40. | —Squirting chloroform into the nose | do. |
| E. | 2 | 49 | 40. | —Evulsion of the nails | do. |
| F. | 2 | 50 | 55. | —Extraction of teeth | do. |
| G. | 2 | 52 | 55. | —Violent slap with open hand on abdomen | do. |
| H. | 2 | 54 | 30. | —Restoration to the recumbent position on the table. | |
| I. | 2 | 56 | 20. | —Inversion of the body so that the dog was completely head downwards and again lowering to the recumbent position. | |
| J. | 2 | 57 | 40. | —Chloroform administration in the ordinary position. | |
| K. | 3 | 1 | 0. | —Attempts to pass grooved staff and Thompson's dilators into the bladder. | |
| L. | 3 | 15 | 5. | —Slitting prepuce. | |
| M. | 3 | 17 | 20. | —Pushing chloroform carelessly until after respiration had ceased. | |

Temperature after death 99 F. The organs did not appear to be fatty.

## November 13th.—No. 80.

Large monkey (Macacus), 21 lbs., caught in the hospital compound. Into chloroform box at 3h. 31m. 38s. On the table and kept quiet with chloroform 3h. 37m. 40s. Temperature 100·2 F. Canula inserted into left carotid. Loop under both vagi. Connection made with manometers at 2h. 39m. 30s.

Two Ludwig and two Fick tracings.

H. M. S. *Observations.*

A. 4 7 40.—Ordinary chloroform administration. The effect of struggling is well shown.

B. 4 11 0.—Irritation of right vagus, entire, coils 8½ cent. distant (Fick 4).

C. 4 14 10.—Irritation of left vagus, entire, 8½ coil (Fick 5).

D. 4 14 50.—Chloroform again.

E. 4 17 45.—Injection of ⅕th grain atropine into peritoneal cavity.

F. 4 19 30.—Irritation of right vagus, coil 8½ again (Fick 6).

G. 4 20 30.—Irritation of entire left vagus, coil 0 (Fick 7).

H. 4 22 30.—Pushing chloroform until respiration stopped completely, and restoration by artificial respiration. The effect of struggling is well brought out in this observation.

(Manometer tracing was interrupted at 4h. 33m. to divide the left splanchnic nerve and started afresh at 4h. 49m. 50s. after cutting the left splanchnic nerve which did not appear to effect pressure much.)

I. 4 50 40.—Administration of chloroform for a short time.

J. 4 53 0.—Evulsion of nails.

K. 4 56 30.—Squeezing and afterwards smashing the testicles.

L. 4 58 20.—Pushing chloroform.

M. 4 59 0.—Thrusting needle into the heart and death.

Slow gasping movements of the chest continued after the heart had ceased to beat. Temperature after death 98·2 F. Post-mortem—There was very little hæmorrhage into the abdomen. The left splanchnic divided, right intact. No clot in heart.

November 13th.—No. 81.

Medium-sized pariah dog, weight 30 lbs., which has been dosed with phosphorus for 2 days, 1-16th of a box of paste each day. Into chloroform box at 7h. 50m. 30s. Fallen down at 8h. 1m. Placed on the table at 8h. 4m. 20s. Carotid ligatured at 8h. 14m. 10s. Canula inserted at 8h. 19m 30s. Temperature

100·4 F. in the rectum. Connection made with manometers at 8h. 25m. 10s. Loops under both vagi. Two Ludwig and one Fick tracing.

| | H. | M. | S. | *Observations.* |
|---|---|---|---|---|
| A. | 8 | 28 | 30. | —Application of bicarbonate of soda to the entire right vagus. |
| B. | 8 | 30 | 35. | —Change to the vertical position and restoration to the recumbent again. |
| C. | 8 | 33 | 20. | —Chloroform administration. |
| D. | 8 | 37 | 10. | —Blow on testicles. |
| E. | 8 | 41 | 0. | —Pushing chloroform with struggling until respiration ceased (*vide* Fick 5 and 6). |
| F. | 8 | 42 | 10. | —Artificial respiration. |
| G. | 8 | 47 | 30. | —Injection of $\frac{1}{5}$th grain atropine into the peritoneum. |
| H. | 8 | 53 | 10. | —Irritation of right entire vagus, coil at 0. |
| I. | 8 | 56 | 0. | —The same (*vide* Fick 9). |
| J. | 8 | 57 | 30. | —Pushing chloroform with violent struggling. |

Fall of pressure very rapid, and the dog died in spite of artificial respiration. A clot in the vessels interrupted the tracing before the heart stopped. Temperature after death 97·4 F. Post-mortem—Organs not particularly fatty.

### November 13th.—No. 82.

Good-sized monkey (Macacus). Weight 15 lbs. Temperature of the room 20·5 Cent. Into chloroform box at 10h. 57m. 18s. Fallen down at 10h. 4m. 17s. On the table at 10h. 6m. 52s. and kept quiet with chloroform. Artery ligatured at 10h. 35m. 53s. Canula inserted at 10h. 39m. 30s. Temperature 99·2 F. in the rectum. Connection made with manometers at 10h 47m. 50s. Two Ludwig and one Fick tracing.

| | H. | M. | S. | *Observations.* |
|---|---|---|---|---|
| A. | 10 | 53 | 30. | —Chloroform administration on a cap in the ordinary way; violent struggling. |
| B. | 10 | 58 | 10. | —Pushing chloroform until respiration stopped; recovery without artificial respiration. |
| C. | 11 | 10 | 20. | —Division of both vagi. |
| D. | 11 | 19 | 10. | —Pushing chloroform until death resulted. |

Temperature after death 99·4 F.

## November 13th.—No. 83.

Medium-sized monkey (Macacus). Weight 12 lbs. Temperature of the room 23 Cent. Into chloroform box at 2h. 21m. 33s. Fallen down at 2h. 30m. 15s. Placed on table at 2h. 32m. 10s. Carotid ligatured at 2h. 39m. 35s. and canula inserted. Temperature in the rectum 103·2 F. Kept quiet with chloroform until connection made with manometers at 2h. 46m. 15s. Three Ludwig and one Fick tracing.

*Observations.*

H. M. S.

A. 2 47 0.—Gradual coming out of chloroform.

B. 2 49 0.—Ordinary chloroform administration with struggling, showing great irregularity of the blood-pressure.

C. 2 52 0.—Change of position to the vertical, during which—

D. 2 53 0.—Extraction of teeth.

E. 2 55 20.—More chloroform and struggling at intervals.

F. 2 58 30.—Restoration to recumbent position.

G. 3 5 0.—Exposure of the ulna nerve at the elbow.

H. 3 7 0.—Tugging at the ulna nerve.

I. 3 8 20.—Incision into abdomen over the stomach.

J. 3 16 0.—Ligature of a glass tube into that organ ; stitching of the abdominal wound.

K. 3 19 20.—Inflation of stomach with air and suddenly allowing the air to escape.

L. 3 25 0.—Re-opening the wound and re-adjusting the stomach tube and re-stitching the abdominal wound.

M. 3 42 40.—Injection of hot water into stomach.

N. 4 0 0.—Injection of one-twelfth grain apomorphine into peritoneum.

O. 4 1 0.—Placing a thermometer in the rectum.

P. 4 2 30.—Artificial respiration at times, the respiration being gasping and irregular.

Q. 4 9 40.—Iced water into stomach : running out through the nose.

Experiment interrupted by hopeless clotting at 4h. 12m. Stopped breathing at 4h. 17m. 40s. The heart-needle stopped moving a few seconds before the thorax was opened and the heart was found to be quite still. Temperature after death 97·8 F.

### November 14th.—No. 84.

Temperature of room 19·75 Cent. Medium-sized, well-nourished pariah dog, weight 35¼ lbs., who has had daily doses of phosphorus for three days. Chloroform commenced in box at 7h. 37m. Fallen down at 7h. 42m. On table at 7h. 44m. 45s. Artery ligatured at 8h. 52m. and canula inserted. Temperature in the rectum 100·8 F. Connection made with manometers at 8h. 10m. 30s. Two Ludwig and one Fick tracing.

| | H. | M. | S. | *Observations.* |
|---|---|---|---|---|
| A. | 8 | 13 | 20. | —Division of rectus muscle of the eye-ball. |
| B. | 8 | 16 | 20. | —Blow on the testicle. |
| C. | 8 | 24 | 0. | —Chloroform administration repeatedly, often accompanied by struggling. |
| D. | 8 | 24 | 40. | —Evulsion of nails. |
| E. | 8 | 25 | 39. | —Snipping anus. |
| F. | 8 | 27 | 0. | —Pushing chloroform until respiration ceased. |
| G. | 8 | 29 | 40. | —Artificial respiration. |
| H. | 8 | 41 | 0. | —Exposure, ligature, and division between ligatures of both vagi. |
| I. | 8 | 45 | 30. | —Pushing chloroform until respiration ceased; fatal result (*vide* Fick 8). |

From the character of Fick reading 8 and the Ludwig tracing just before, it is doubtful if the vagi were really divided. Temperature after death 97 F.

In this experiment the absence of any effects from the operations which are usually said to be dangerous under chloroform is well brought out; also the rapid and irregular falls of the blood-pressure when the animal struggles and breathes irregularly during the inhalation of the anæsthetic.

### November 14th.—No. 85.

Temperature of the room 21 Cent. Medium-sized dog, weight 30¼ lbs., that has had phosphorus daily for 3 days, 1-16th of a box. Into chloroform box at 9h. 42m. Fallen down at 9h. 45m. 10s. Placed on the table at 9h. 46m. 45s., after which its breathing was noticed to have stopped. Artificial respiration was commenced at once, but failed to bring him round, although there was a pulse some time after it commenced.

This is a case where a weak sick animal was left in the chloroform box too long, and its respiration neglected while it was being transferred to the table.

### November 14th.—No. 86.

Monkey (medium-sized Macacus). Weight 11 lbs. Into chloroform box at 10h. 27m. 22s. Fallen down at 10h. 13m. 15s. Placed on the table and kept quiet with chloroform. Carotid ligatured at 10h. 33m. 40s. Canula inserted at 10h. 39m. 30s. Temperature in the rectum 101 F. Connection made with the manometers at 10h. 44m. Three Ludwig and one Fick tracing.

*Observations.*

H. M. S.

A. 10 45 20.—Small incision into abdomen.

B. 10 47 10.—Administration of chloroform : struggling, and irregularity of the blood-pressure.

C. 10 49 50.—Injection of 1-12th grain apomorphine into abdominal cavity. (The animal then lay almost perfectly quiet without more chloroform for almost an hour, during which he was given another dose of apomorphine 1-12th grain.)

D. 11 43 30.—Pushing chloroform, with struggling, until respiration ceased ; recovery with artificial respiration.

E. 11 46 0.—Thrusting a needle into the chest but not into the heart.

F. 11 51 30.—Effect of letting the tongue drop.

G. 11 54 30.—Repetition of D and E, and thrusting a needle into the heart.

H. 12 19 0.—Injection of atropine, ¼ grain, in abdomen, and finally pushing chloroform until death resulted.

Thorax opened at 12h. 26m. respiration having stopped, but afterwards there were again regular movements of the diaphragm, which continued off and on until 12h. 23m. 50s., when they finally stopped. The tracing was stopped by hopeless clotting at 12h. 31m. Heart stopped very gradually, but not finally until 12h. 37m. Temperature after death 95·4 F.

November 14th.—No. 87.

Mr. Ulett's bull dog, weight 31 lbs., suffering from tumours in the inguinal region. Into chloroform box at 2h. 14m. 10s. Fallen down at 2h. 23m. 10s. Placed on the table and kept quiet with chloroform. Temperature in the rectum 102 F. Loops under both vagi. Artery ligatured at 2h. 34m. 30s. Canula inserted 2h. 40m. 20s. Connection made with manometers at 2h. 43m. 40s. One Ludwig and one Fick tracing.

| | H. | M. | S. | *Observations.* |
|---|---|---|---|---|
| A. | 2 | 46 | 30. | —Ordinary chloroform administration. |
| B. | 2 | 48 | 0. | —Irritation of entire right vagus with coils at 5 centimetre distance. |
| C. | 2 | 53 | 50. | —Pushing chloroform, with accidental compression of the neck by a strap (*vide* Fick 3). The same slowing of the pulse was produced as is observed when the vagus is irritated. |
| D. | 3 | 0 | 0. | —Pushing chloroform until respiration completely ceased.<br>Failure to restore the animal by artificial respiration. |

Temperature after death 101·2 F.

November 14th.—No. 88.

Temperature of room 24 Cent. Medium-sized pariah dog. Weight 30 lbs. Into chloroform box at 3h. 22m. 26s. Fallen down at 3h. 24m. 45s. Placed on the table at 3h. 26m. 25s. Artery ligatured at 3h. 37m. Temperature in the rectum 100·2 F. Canula inserted at 3h. 40m. 53s. Connection made with manometers at 3h. 50m. 11s. Loops under both vagi. One Ludwig and one Fick tracing.

| | H. | M. | S. | *Observations.* |
|---|---|---|---|---|
| A. | 3 | 51 | 0. | —Pushing chloroform accompanied by struggling until respiration stopped. (The trace is spoilt by the drum not rotating for nearly a minute.) The fall of pressure was extremely rapid and irregular. Fick 2 shows that when the respiration is stopped by an over-dose of chloroform, a condition of pulse similar to that caused by vagus stimulation may be produced. |

H. M. S.

B. 3 52 50.—Recovery with artificial respiration.

C. 3 59 20.—Pressure on abdomen.

Pushing chloroform again until respiration stopped.

D. 4 1 0.— Division of both vagi while the animal was deeply under. Fick 5 shows that after division of the vagi the slowing of the pulse observed in stoppage of the respiration in chloroform poisoning did not occur.

E. 4 4 0.—Gradual fall of pressure and death in spite of artificial respiration.

Temperature after death 98·4 F.

November 15th.—No. 89.

Temperature of the room 21 Cent. Unusually large brindled dog. Weight 88 lbs. Into chloroform box at 7h. 38m. Fallen down at 7h. 46m. 20s. Placed on the table 7h. 48m. 50s. Temperature in the rectum 99·8 F. Left carotid ligatured at 8h. 2m. 5s. Connection made with manometer at 8h. 6m. 30s. Four Ludwig and two Fick tracings.

*Observations.*

H. M. S.

A. 8 10 45.—Chloroform administration with struggling (the excursions of the marker had previously been unusually great), and pushing the same until respiration ceased.

B. 8 24 20.—Artificial respiration and gradual recovery.

C. 8 29 30.—Division of both vagi.

D. 8 38 10.— { Administration of chloroform again until respiration ceased, and Artificial respiration as before.

E. 8 47 40.—Compression of the chest and administration of chloroform on a saturated sponge at the moment the chest was released.

F. 8 51 0.—Pressure on abdomen.

H. M. S.

G. 8 51 0.—Pushing chloroform again until respiration stopped: and artificial respiration as before.

H. 8 55 40.—Tracheotomy and insertion of a large glass tube into the trachea.

I. 8 59 0.—Repetition of F by stopping the tracheal tube when the chest was in the expiratory position, holding chloroform in front of the tube, and then suddenly opening it.

J. 9 5 40.—Continued administration of chloroform by holding a sponge or towel before the end of the tube.

K. 9 7 15.—Closing the tracheal tube so as to cause asphyxia.

L. 9 12 25.—Administration of chloroform by Junker's tube introduced into the tracheal tube, which remained open, and pushing it again until respiration stopped, and artificial respiration as before.

M. 9 26 10.—Administration of ether in the same way, but this failed to keep the animal thoroughly under. After chloroform had been again given the manometer was disconnected in order to divide the medulla oblongata.

All attempts to reconnect after its division failed on account of clot in the vessel, and the heart ceased beating at 11h. Temperature at 11h. 5m., 96 F. in the rectum.

In this experiment slowing of the pulse, in Observations A and B, which occurs when the respiration fails from over-dosing with chloroform is shown (*vide* Fick 6 and 9). This effect is not produced after division of the vagi (*vide* Fick 10 and 11). The effect of asphyxia in lowering the blood-pressure is also seen in the Ludwig tracing at 9h. 7m. 20s. and 9h. 9m. 40s. The difference in intensity between diluted chloroform and diluted ether, administered in precisely the same way, is brought out in Observations L and M.

## November 15th.—No. 90.

Medium-sized strong pariah, given 1 grain of morphine hypodermically at 2h. 24m. 10s. Temperature of the room 24½ Cent. Salivated a good deal at

2h. 33m. and afterwards went to sleep. At 4h. 15m. he was still irritable and unfit for experiment.

### November. 15th.—No. 91.

Large pariah dog, 38 lbs. Temperature of the room 24·5 Cent. Chloroform given at 2h. 34m. 45s.. Fallen down at 2h. 41m. 30s. On the table at 2h. 43m. 15s. and kept quiet with chloroform. Temperature in the rectum 100·4 F. Right jugular vein ligatured at 2h. 51m. Canula into carotid at 2h. 58m. 5s. Connection made with manometers at 3h. 1m. 30s. The pressure in the manometer had been arranged a little too high before commencing, so that some bicarbonate of soda must have been shot into the vessel. No convulsions occurred. Three Ludwig and one Fick tracing.

*Observations.*

H. M. S.

A. 3 6 0.—A long period in which the dog was left without chloroform and occasionally struggled.

B. 3 35 20.—Injection at different times of 5 doses of chloroform, 20 minims each (except in one instance when about 5 minims or possibly more was spilled), into the jugular vein. After the 5th dose the dog died, the heart stopping at 4h. 13m. Temperature in rectum at death 97·2 F. Post-mortem.—No clots in heart.

### November 18th.—No. 92.

Temperature of the room 23 Cent. Large-sized pariah dog. Weight 33¼ lbs. rather thin. Into chloroform box at 7h. 24m. 43s. Fallen down at 7h. 28m. 53s. On the table at 7h. 29m. 37s. Artery ligatured at 7h. 41m. 8s. and canula inserted. Temperature in the rectum at 7h. 42m. 30s., 100·6 F. Jugular vein tied at 7h. 51m. 47s. Connection made with the manometers at 7h. 58m. 20s. Three Ludwig and one Fick tracing during repeated injections of chloroform into jugular vein. Nine doses were given in the course of an hour, and death ensued very gradually. Temperature after death 97 F. Post-mortem—After the chest was opened 5 or 6 regular movements of opening and shutting the jaws coincident with rhythmical contractions of the diaphragm occurred. The heart was quite still in all its cavities. There were no clots in the heart. No sign of the needle in the heart.

*Observations.*

H. M. S.

A. 8 0 15.—Twenty minims of pure chloroform injected into the jugular vein.

B. 8 2 40.—Repeat.

C. 8 3 50.—Repeat.

D. 8 5 55.—Repeat. This injection was followed by lowering of the blood-pressure, which never rose again. The injection of twenty minims of chloroform was repeated at 8h. 13m. 20s. ; 8h. 14m. 10s. ; 8h. 40m. 30s. ; 8h. 43m. 20s.; and 8h. 46m. 10s. It seems clear from experiment No. 92 that direct action of chloroform upon the heart substance is not the cause of the fall of blood-pressure that occurs when it is inhaled. Repeated injections of twenty minims of pure chloroform were made into the jugular vein, and the effect was not to paralyse the heart, but to produce narcosis with a gradual fall of pressure exactly as if the chloroform had been inhaled. Compare with Observation D of Experiment No. 64.

November 18th.—No. 93.

Temperature of the room 24 Cent. Large pariah dog. Weight 33¼ lbs. Into chloroform box at 9h. 59m. Fallen down at 10h. 5m. 25s. On the table at 10h. 8m. 27s. and kept quiet with chloroform. Artery ligatured at 8h. 20m. 50s. Canula inserted at 8h. 22m. 22s. Temperature in rectum 99·8 F. Connection made with manometer 10h. 28m. 45s. Two Ludwig and one Fick tracing during injection of ether into the jugular vein with at the same time injection of solution of hydrochloric acid (0·8 per cent.) in normal saline solution into the femoral vein. Notice also effect of thrusting needle into heart. Temperature 101·2 F. after death. Post-mortem—Heart very much distended. Right ventricle full of clot. Left free. Superior and inferior vena cava and pulmonary artery filled with clot.

H. M. S. *Observations.*

A. 10 30 10.—Ether on an ordinary cap until 10h. 39m. 40s. The animal was quiescent, but complete narcosis was not produced. A great deal of frothy fluid accumulated in the trachea.

H. M. S.

B. 10 40 20.—Twenty minims of ether injected into the jugular vein. The injection was repeated at 10h. 41m. 10s. and 10h. 42m. 15s. and each time the blood-pressure fell and rose again in 40 or 50 seconds. At 10h. 47m. 10s. and again at 10h. 52m. 30s. forty minims of ether were injected. There was on each of these occasions a very rapid and prolonged fall of blood-pressure and a slow weak pulse with very difficult recovery.

C. 11 1 30.—Injection of sixty minims of ether into the jugular vein. The fall of blood-pressure was immediate and rapid, and the pulse tracing became almost imperceptible (*vide* Fick 6 and 7). The pressure remained low until 11h. 5m. 20s., when slight recovery took place (*vide* the Ludwig tracing at 11h. 5m. 0s.) At 11h. 6m. 0s. sixty more minims of ether were injected into the jugular vein and this injection caused stoppage of the respiration and death. Post-mortem gasps occurred as in Experiment No. 92. The heart was found full of venous clot.

### November 18th.—No. 94.

The dog in No. 90 having escaped from the cage, another dog was given 2 grains of morphia at 8-20 a.m. At 10 o'clock he was still irritable and unfit for experiment, for, although sleepy and at first sight apparently insensitive, he could be easily aroused by even painless stimuli and then ran about in an excited wild way.

### November 18th.—No. 95.

Temperature of the room 25½ Cent. Monkey (medium-sized Macacus). Held down on table and given ether on a cap at 2h. 50m. 45s., his temperature in rectum being 102·4 F. at that time. Ether stopped at 2h. 51m., the animal being quite quiet, though cornea not insensitive. Respiration chiefly abdominal, 68 per minute. Artery ligatured at 3h. 7m. 45s., and loop placed under left vagus. Canula inserted into artery at 3h. 11m. 33s. Left jugular accidentally cut; so ligatured. Loop under right vagus and right carotid. Ether given now and again during above proceedings. Connection made with manometer at 3h. 26m. 50s. Two Ludwig and three Fick tracings.

| | H. | M. | S. | *Observations.* |
|---|---|---|---|---|
| A. | 3 | 26 | 50. | —The quiescent condition, not anæsthesia, produced by ether. |
| B. | 3 | 28 | 10. | —Irritation of the nostril by chloroform, and |
| C. | 3 | 29 | 15. | —Ammonia. |
| D. | 3 | 36 | 5. | —Clamping of the right carotid so as to cut off both carotids. |
| E. | 3 | 35 | 0. | —Extraction of teeth with the key. (*Vide* Fick reading marked "6" on the Fick tracing, but noted as "5" on the Ludwig tracing.) |
| F. | 3 | 50 | 10. | —Exposure and irritation of the sciatic nerve. |
| G. | 3 | 53 | 30. | —Irritation of the entire right vagus coil 10 producing coughing (Fick 8). |
| H. | 3 | 54 | 40. | —Irritation of the entire left vagus coil 10 (Fick 9). |
| I. | 3 | 55 | 50. | —Irritation of the entire right vagus coil 5 (Fick 10). (The effect of irritation of the right was less than that of the left vagus). |
| J. | 4 | 0 | 30. | —Ligature and division of the right vagus (Fick 11). |
| K. | 4 | 14 | 55. | —Irritation of central right vagus coil 5 (Fick 14). |
| L. | 4 | 17 | 15. | —Irritation of peripheral right vagus coil 5 (Fick 15). |
| M. | 4 | 20 | 0. | —Ligature and division (Fick 16) of left vagus. |
| N. | 4 | 21 | 30. | —Irritation of central left vagus coil 5 (Fick 17). |
| O. | 4 | 25 | 0. | —Irritation of peripheral left vagus coil 5 on two occasions (Fick 18, 19), but doubtful if the nerve was really irritated in the first. |
| P. | 4 | 37 | 0. | —Pushing ether on cap closely applied over muzzle until death ensued. |

Manometer stopped on account of clot at 4h. 45m. Needle into heart; hardly moving. Thorax opened 4h. 46m. 20s. Heart still.

No clots in the heart cavities. Temperature at 4h. 35m. 40s. in the rectum 100·2 F.

### November 19th.—No. 96.

Temperature of the room 23 Cent. Medium-sized female monkey (Maçacus). Weight 11¼ lbs. Into chloroform box at 9h. 23m. Fell down at 9h. 31m. On the table at 9h. 33m. Temperature in rectum 100·4 F. Artery ligatured at 9h. 52m. 50s. Canula inserted at 9h. 58m. Connection made with manometer at 10h. 6m. 40s. Two Ludwig and one Fick tracing.

H. M. S. *Observations.*
A. 10 8 20.—Ordinary chloroform administration.
B. 10 16 40.—Bandaging the abdomen.
C. 10 21 0.—Bandaging the lower chest with plaster of Paris.

Death ensued from asphyxia and in spite of artificial respiration after removal of the bandage. Post-mortem—Heart cavities firmly contracted after death, and lungs engorged. Trachea full of froth. Temperature at end of Experiment 96·2 F. in rectum.

### November 19th.—No. 97.

Medium-sized female monkey. Weight 9¾ lbs. Temperature of the room 23½ Cent. Into chloroform box at 10h. 50m. Fallen down at 10h. 58m. 30s., and placed on the table. Temperature in rectum 102·4 F. Artery ligatured at 11h. 11m. 10s., and canula inserted. Connection made with manometers at 11h. 24m. 50s. One Ludwig and one Fick tracing.

H. M. S. *Observations.*
A. 11 26 30.—Struggling.
B. 11 27 30.—Application of a plaster of Paris bandage to the chest to imitate stays, and of
C. 11 32 0.—A tight broad tape round the lower part of the abdomen to imitate the effect of petticoats.
D. 11 33 0.—Pushing chloroform until respiration stopped.

Death ensued although the bandages were quickly removed and in spite of artificial respiration. Post-mortem—Blood, arterial, in left side of heart. Temperature after death 102·4 F.

### November 19th.—No. 98.

Small monkey fastened on to the rabbit board at 3h. 14m. 30s. A pin placed in the heart and connected by a thread with a time-marker writing on a quick revolving drum (the same as is used for the Fick manometer). The experiment was imperfect, but a few readings are preserved showing effect of placing ammonia before the nose.

November 20th.—No. 99.

Small rabbit on to rabbit board at 7h. 30m. with a needle in its heart attached by a long thread to a time-marker. The trace shows the effect of holding ammonia before the nose.

November 20th.—No. 100.

A monkey arranged in the same way. One trace showing the absence of any effect when ammonia is held before the nose, corresponding to that which occurs in the rabbit.

November 20th.—No. 101.

Small rabbit arranged as above and chloroform held before his nose. One tracing incorporated with the first tracing of 102.

November 20th.—No. 102.

Another rabbit in the same way. Chloroform given as before. Two tracings.

November 20th.—No. 103.

Large monkey. Weight 16¼ lbs. Temperature of the room 24 Cent. Into chloroform box at 9h. 8m. 44s. Fallen down at 9h. 15m. 5s. Placed on the table at 9h. 19m. Temperature in rectum 101·8 F. Artery ligatured at 9h. 37m. 10s. Canula inserted at 9h. 40m. 30s. Connection with manometer at 10h. 3m. 15s. Four Ludwig tracings and three Fick.

*Observations.*

|  | H. | M. | S. |  |
|---|---|---|---|---|
| A. | 10 | 6 | 20. | —Mechanical irritation of the left vagus. |
| B. | 10 | 7 | 30. | —Struggling. |
| C. | 10 | 8 | 20. | —Irritation of the entire right vagus, coils 10 Cent. apart (Fick 4). |
| D. | 10 | 14 | 55. | —Irritation of the entire left vagus (Fick 5). |
| E. | 10 | 17 | 30. | —Irritation of the left vagus and pushing chloroform at the same time. |

H. M. S.

F. 10 24 40.—Ordinary chloroform administration.

G. 10 27 15.—Extraction of teeth (Fick 10).

H. 10 42 0.—Blow on the abdomen with a fly-flapper (Fick 13).

I. 10 49 10.—Attempts to pass a grooved staff into bladder.

K. 10 54 0.—Castration.

L. 11 5 0.—Electrical irritation of the spermatic cord.

M. 11 5 40.—Exposing the femoral vessels.

N. 11 16 20.—Transfusion of saline solution, about 220 c.c., into the femoral vein.

O. 11 26 0.—Exposing and destroying the left splanchnic nerve and plexus, during which operation the aorta was accidentally snipped, but the opening was at once closed by a clamp and very little hæmorrhage occurred.

P. 11 39 10.—Thrusting a needle into the heart.

Q. 11 43 0.—Pushing chloroform until death with attempted restoration by means of transfusion of saline solution and artificial respiration.

After the thorax was opened and the heart found to have quite ceased beating, rhythmical movements of the diaphragm occurred. The heart continued irritable for some minutes. Temperature after death 97·2 F.

In Experiment 103, as well as in Experiments 80 and 92, rhythmical movements of the diaphragm were noticed after the heart had ceased beating and after the chest had been opened. In Experiment 103 the splanchnic nerve had been divided, death and stoppage of the heart were very gradual, and the heart was still irritable during the diaphragmatic contractions.

## November 20th.—No. 104.

A dog, weight 36½ lbs., which had been dosed with phosphorus for two days. Into chloroform box at 2h. 22m. 5s. Fell down at 2h. 26m. 35s. On the table at 2h. 29m. 30s., and kept quiet with chloroform. Temperature in rectum 100·4 F. Artery ligatured at 2h. 34m. 7s. Canula inserted at 2h. 36m.

7s. Connection with manometer at 2h. 40m. 45s. Three Ludwig and three Fick tracings.

*Observations.*

H. M. S.

A. 2 41 0.—Struggling at various times.

B. 2 41 30.—Chloroform administration; violent blow on the testicle at 2h. 41m. 50s. No shock.

C. 2 42 30.—Slight embarrassment of the respiration, from the foreleg being tightly bound down to the board (*vide* Fick 1 & 2).

D. 2 48 40.—Pushing chloroform until respiration stopped. During this observation the animal held his breath, the pulse tracing was immediately lost and the pressure fell exactly as in the Glasgow experiments A & C.

E. 2 51 10.—Artificial respiration (*vide* Fick 5), when the normal respiration was recommencing. The slow pulse is very noticeable in this observation.

F. 2 53 50.—Change to the vertical position and while vertical more blows on the testicle, and dental operations were performed, without the production of shock.

G. 3 2 40. —Incising the abdomen.

H. 3 3 30.—Blowing apomorphia 1-12th grain in solution into the peritoneal cavity; vomiting (Fick 9).

I. 3 23 0.—Small opening into the larynx and injection of a few drops of chloroform.

J. 3 26 30.—Pushing chloroform again until respiration ceased and artificial respiration (*vide* Fick 12).

K. 3 34 40.—Seizing and snipping the skin of the anus.

L. 3 35 40.—Larger opening into abdomen and tying a tube into the stomach.

M. 3 41 40.—Distension of the stomach with air.

N. 3 49 55.—Pulling the stomach forcibly.

O. 3 52 10.—Pushing chloroform very far, and prolonged artificial respiration.

H. M. S.
P. 3 59 10.—Electrical stimulation of the heart.
Q. 4 5 50.—Placing ice on the abdominal cavity.
R. 4 9 0.—Pushing chloroform again until death resulted.

After the thorax was opened 37 rhythmical contractions of the diaphragm occurred, but the heart continued beating for more than two minutes after these had stopped. Post-mortem—Temperature 98·2 F. Weight post-mortem 34¾ lbs.

### November 21st.—No. 105.

Large-sized pariah dog, weight 30¼ lbs., which has had three doses of phosphorus previously on the 18th, 19th, and 20th instant, respectively. It is in consequence very much out of sorts, and, judging from previous experiments with other dogs, would certainly have died in a few hours. Into chloroform box at 7h. 44m. 25s. Fallen down at 7h. 48m. 55s. Placed on the table and a canula inserted into left carotid. Temperature in rectum 102 F. Connection made with manometers at 8h. 9m. 10s. One Ludwig and one Fick tracing.

*Observations.*

H. M. S.
A. 8 12 50.—Pushing chloroform until respiration ceased (*vide* Fick 2 and 3).
B. 8 16 50.—Recovery by artificial respiration.
C. 8 22 35.—Snipping skin of the anus (*vide* Fick 5). No sign of shock.
D. 8 24 30.—Ordinary chloroform administration (trace imperfect owing to the drum sticking).
E. 8 28 55.—Pushing chloroform again and death in spite of artificial respiration.

The trachea was for some reason considerably obstructed with fluid, so that the animal was probably in a state of partial asphyxia. Temperature at death 100·8 F.

### November 21st.—106.

Large-sized pariah. Weight 25¾ lbs. Has had three daily doses of phosphorus on 18th, 19th, and 20th evenings. Given chloroform at 9h. 6m. Fallen down at 9h. 15m. 30s. On the table at 9h. 16m. Artery ligatured at

9h. 25m. 30s. Temperature in rectum 100·2 F. Canula inserted at 9h. 29m. 30s. Connection made with manometer at 9h. 41m. One Ludwig and one Fick tracing.

H. M. S. *Observations.*

A. 9 42 20.—Ether administration on cap.

B. 9 46 10.—Pushing ether until respiration stopped. Fick 4 shows exactly the same absence of pulse tracing which is noted so frequently in chloroform poisoning when the respiration stops (*vide* Experiment No. 107, Fick 3, and Experiment No. 178—all the tracings).

C. 9 52 0.—Artificial respiration ; and

D. 9 54 30.—Inversion of the body failed to restore the animal.

Temperature after death 97·8 F. Post-mortem.—There was a severe rupture in the liver and a quantity of blood in the peritoneum. Liver distinctly soft, friable and pale in colour.

Experiment No. 106 shows the effect of inversion of the body, which is now-a-days thought to be a restorative in chloroform poisoning. Inversion of the body causes the pressure in the carotid to rise, but it falls to its former state when the animal is replaced in the horizontal position. In Observation D at 9h. 54m. 30s., when the animal was in the last stage of ether poisoning, inversion of the body raised the pressure in the usual way as long as it was continued ; but it failed to restore the respiration and the dog died. The change in the blood-pressure in the carotid, which occurs when the position of the body is changed, appears therefore to be due simply and solely to the effect of gravity. Experiment No. 106 also proves that the effect of ether is precisely the same as that of chloroform only less intense.

### November 21st.—No. 107.

Large-sized pariah. Weight 28lbs. Has had two doses of phosphorus on the 18th and 19th instant, respectively. Into chloroform box at 10h. 9m. 14s. Fallen down at 10h. 14m. 27s. On the table at 10h. 17m. 30s. Artery ligatured at 10h. 24m. 13s. and canula inserted. Temperature, 99·8 F. in rectum. Loop under both vagi and the right carotid. Connection made with manometers at 10h. 38m. Pressure much too high ; so much soda solution must have been shot into the artery (*vide* Ludwig tracing).

One Ludwig and one Fick tracing.

H. M. S. *Observations.*

A. 10 36 0.—Coming out of chloroform.

B. 10 43 20.—Mild irritation (coil 15) of the entire right vagus (Fick 2).

C. 10 44 20.—Pushing chloroform until respiration stopped (*vide* Fick 3), pulse tracing absent, this not being a sign that the heart had stopped ; it was restored easily by artificial respiration.

D. 10 46 0.—Recovery with artificial respiration.

E. 10 51 0.—Ligature and division of the right vagus (Fick 5).

F. 10 53 10.—Mild irritation (coil 15) of the peripheral end of the right vagus (Fick 6). Pushing chloroform again during struggling until death occurred in spite of artificial respiration.

The temperature in rectum was noted at intervals throughout (*vide* Ludwig tracing).

November 21st.—No. 108.

Large pariah. Weight 26 lbs. Has had two one-grain pills of phosphorus on the 18 and 19th instant, respectively. Into chloroform box at 11h. 23m. Fallen down at 11h. 27m. 45s. On the table at 11h. 30m. 25s. and kept quiet with ether. Temperature 100·8 F. in rectum at 11h. 33m. Artery ligatured at 11h. 37m. 45s. Canula inserted at 11h. 41m. 30s. Temperature 100·6 F. at 11h. 47m. 15s. One Ludwig and one Fick tracing.

H. M. S. *Observations.*

A. 11 57 40.—Continued ether administration, which failed for a long time to keep the cornea insensitive, and was only effective when the animal was half smothered.

B. 12 0 40.—Mild irritation of the entire right vagus, coil 15 (Fick 2).

C. 12 2 0.—Convulsion of the whole body.

D. 12 10 30.—Irritation of right vagus, coil 10 (Fick 6). This reduced the pulse from 120 to 37 a minute.

E. 12 14 40.—Ligature and division (Fick 7) of the right vagus.

F. 12 16 0.—The ether was pushed until death ensued.

Tracing stopped at 12h. 21m. Heart still moving when the thorax was opened at 12h. 25m. Temperature 97·4 F. Heart flickering at 12h. 27m.

### November 22nd.—No. 109.

Medium-sized pariah, weight 27 lbs., that has had three doses of phosphorus on the 18th, 19th, and 20th instant, and is in an extremely feeble state. Chloroformed in the box at 8h. 9m. Fallen down at 8h. 17m. 15s. Placed on the table at 8h. 19m. 20s. Noticed to have stopped breathing at 8h. 19m. 40s. Artificial respiration begun at once. Needle in the heart moving feebly at 8h. 21m. 45s. Breathing spontaneously at 8h. 23m. 20s., but artificial respiration continued in the ordinary way. Respiration continued off and on until 8h. 26m. 30s. Needle in the heart afterwards moving slightly, but rhythmically. The trachea was opened and artificial respiration performed by the bellows method for a few minutes, but with no result.

### November 22nd.—No. 110.

Medium-sized female monkey, weight 11¾ lbs. (has varicose veins on abdomen). Into chloroform box at 8h. 51m. Fallen down at 8h. 54m. 15s. On the table at 8h. 56m. 45s. Temperature in rectum 101·4 F. Artery ligatured at 9h. 10m. 5s. Canula inserted 9h. 12m. 45s. Loops under both vagi, Breathing stopped at 9h. 23m. 50s. Artificial respiration at once and until 9h. 24m. 50s. when breathing became natural again. Stopped breathing again at 9h. 27m. and artificial respiration performed for some time but without success. Temperature after death 100·8 F. Post-mortem—The large bronchi and trachea were filled with blood, and there was a large patch of bloody effusion in the posterior lobe of one of the lungs.

### November 22nd.—No. 111.

Monkey, weight 10¾ lbs., chloroformed in the box 9h. 43m. 50s. Monkey fallen down at 9h. 48m. 45s. On the table 9h. 50m. 40s. Artery ligatured and canula inserted. Connection with manometer at 10h. 13m. 30s. Four Ludwig and two Fick tracings.

H. M. S. *Observations.*

A. 10 14 30.—Ordinary chloroform administration on several occasions.

B. 10 20 40.—Small incision into the abdomen and tying a small tube into the colon (erroneously written "stomach" on the tracing).

H. M. S.

C. 10 51 0.—Injection of two doses of half a grain of tartar emetic into the colon; struggling.

D. 11 15 0.—Exposing and destroying the left splanchnic cord and plexus, during which proceeding the animal was detached from the manometer.

E. 11 34 20.—Pushing chloroform until respiration had almost stopped.

F. 12 8 20.—Application of hot and afterwards cold cloths to the body.

G. 12 14 40.—Placing a clean cap over the nose.

H. 12 12 30.—Holding ammonia before the nose.

I. 12 20 0.—Pushing chloroform until respiration ceased.

J. 12 27 0.—Artificial respiration.

K. 12 31 20.—Exposing the jugular vein.

L. 12 36 0.—Injection of ammonia, 5 minims of strong ammonia diluted with 20 minims of water into the jugular.

M. 12 40 0.—Pushing chloroform finally until death resulted. The decline of blood-pressure was unusually gradual. The temperature in the rectum had fallen below 95 F. for some time before the end of the experiment.

*N.B.*—Readings marked " C " were taken with a second Fick instrument made by the Cambridge Co.

To test the effect of shock due to vaso motor change rather than affection of the heart Goltz's experiment on the frog was repeated on three dogs. In one there was slight lowering of pressure which was not extensive, and in the others no effect was produced at all. Other operations, which seemed likely to produce shock, such as violent blows upon the testicle, were singularly devoid of effect. Failing to lower the blood-pressure by any of these methods, recourse was had to section of the splanchnics, but the low condition of blood-pressure this produced appeared, like stoppage of the heart from vagus irritation, to be a source of safety rather than of danger during chloroform administration. In this connection Experiment No. 111 may be studied. There was not much external hæmorrhage, but the splanchnics were divided,—a proceeding which, as is often said, bleeds the animal into his own vessels. The pressure was after this extremely low, but chloroform was repeatedly given, and various other actions taken, and then chloroform had to be pushed on a saturated sponge enclosed in a cap for eleven minutes before respiration ceased.

### November 22nd.—No. 112.

Temperature of the room 24½ Cent. Into chloroform box at 2h. 41m. 19s. Fallen down at 2h. 44m. 47s. On the table at 2h. 46m. Observed to have stopped breathing at 2h. 46m. 10s. Artificial respiration until he was pronounced "dead" at 3h. 2m. Needle in the heart faintly moving at 2h. 54m. 30s. Post-mortem showed all the signs of asphyxia.

There were thus three accidental deaths in the course of this one day, November 22nd, which it may be noted was exceptional in being a wet day and was characterised by constant blunders and mistakes committed by every one engaged in the experiments and in every department (*vide*, for example, the blunders and smudges on the tracings in No. 111) as well as in the administration of chloroform.

### November 22nd.—No. 113.

Temperature of the room 24½ Cent. Pariah dog. Weight 38 lbs. Into the chloroform box at 3h. 5m. 30s. Placed on the board at 3h. 8m. 10s. and kept quiet with chloroform. Artery ligatured at 3h. 19m. 10s. Canula inserted at 3h. 23m. 40s. Temperature in the rectum 100·4 F. at 3h. 23m. 45s. Connection with manometer at 3h. 30m. Three Ludwig and two Fick tracings.

| | H. | M. | S. | *Observations.* |
|---|---|---|---|---|
| A. | 3 | 30 | 0. | —Chloroform administration and struggling (the trace is imperfect on account of the drum sticking). |
| B. | 3 | 32 | 20. | —Cessation of respiration and artificial respiration. |
| C. | 3 | 33 | 25. | —Division of the right and then the left vagus. |
| D. | 3 | 38 | 0. | —Pushing chloroform again until respiration stopped and restoration by artificial respiration. |
| E. | 3 | 39 | 30. | —Exposing the femoral vessels and repetition of (D). |
| F. | 3 | 51 | 0. | —Ligature of femoral artery and vein. |
| G. | 4 | 3 | 10. | —Hæmorrhage to 170 cc. from the femoral artery and chloroform again. |
| H. | 4 | 14 | 20. | —Transfusion of saline solution into femoral vein 250 cc. in two doses. |

H. M. S.
I. 4 20 30.—Pushing chloroform again as in D.

J. 4 26 55.—Hæmorrhage again 250 cc. of blood taken.

K. 4 31 0.—Repetition of the transfusion and pushing chloroform as before.

Failure to restore the animal in spite of artificial respiration and transfusion of ammonia. Temperature in the rectum gradually fell to 96·8 F. at death. Post-mortem.—Right heart filled with coagula.

November 23rd.—No. 114.

Rabbit into cubic foot box with paper saturated with chloroform at 7h. 41m. 10s. Breathing, which was held at first, rapid at 7h. 42m. Fallen on its side at 7h. 42m. 30s. Breathing stopped at 7h. 42m. 50s. Stopped chloroform and performed artificial respiration at 7h. 43m. Convulsions at 7h. 44m. Pupils gradually dilating. Convulsions nearly ceased at 7h. 45m. Came round at 7h. 46m. No connection was made with the recording apparatus, and there is therefore no tracing.

November 23rd.—No. 115.

Monkey. Weight 9¾ lbs. Temperature of the room 23 Cent. Into chloroform box at 7h. 49m. 10s. Fallen down at 7h. 52m. 10s. Placed on the board at 7h. 53m. and kept quiet with chloroform. Left carotid exposed and a loop placed under the right vagus. Operation to expose the splanchnics commenced at 8h. 11m. 30s. Left splanchnic ligatured at 8h. 18m. and the plexus excised. Artery ligatured at 8h. 24m. 5s. Canula inserted at 8h. 29m. 10s. Temperature in rectum 97 F. Connection with manometer at 8h. 37m. 40s. One Ludwig and one Fick tracing.

*Observations.*

H. M. S.
A. 8 38 45.—Administration of chloroform ; struggling.

B. 8 44 0.—Administration of ether on a cap.

C. 8 46 50.—Asphyxia from the trachea becoming obstructed with fluid.

D. 8 59 10.—Introduction of needle into the heart.

E. 9 2 50.—Pulling and cutting the right vagus.

The temperature in the rectum is noted from time to time on the Ludwig tracings. At 9h. 5m. the experiment was brought to an end by the canula cutting through the artery, and eventually the animal was killed with chloroform.

(After the manometer experiment was closed the convolutions of the brain were exposed, but stimulation failed to give any result.)

### November 23rd.—No. 116.

Monkey, weight 12½ lbs., given two grains of morphine under the skin of the thigh at 10h. 1m. Tied on to the dog-board at 10h. 19m. and two grains more morphine injected at 10h. 20m. 10s. Quite wide awake at 10h. 30m. 10s. Given a little chloroform at 10h. 35m. 30s., when he was slightly under the influence of morphine and kept quiet with chloroform. Loop under both vagi. Right pupil slightly dilated. Left contracted at 10h. 48m. Artery ligatured at 10h. 50m. 45s. Canula inserted at 10h. 53m. 15s. Right pupil then widely dilated. Temperature 100·6 F. Connection with manometer at 11h. 8m. Two Ludwig and two Fick tracings.

*Observations.*

H. M. S.

A. 11 12 30.—Ordinary chloroform administration.

B. 11 17 0.—Ligature of the femoral artery and vein.

C. 11 29 25.—Hæmorrhage from femoral artery.

D. 11 31 10.—Transfusion of saline solution and ammonia (5 minims liquor ammoniæ fort. to 25 ounces of saline). Respiration stopped at 12h. 11m. 30s., but after continuous artificial respiration the animal gave one gasp at 12h. 22m. 30s. and began breathing naturally at 12h. 23m. 30s. During almost the whole of this time the manometer tracing was interrupted by clots in the tubes.

E. 11 55 0.—Ether inhalation at short intervals.

F. 12 14 30.—Needle into the heart.

G. 12 30 35.—Small incision into abdomen, and injection of spirits of ammonia into the stomach.

H. 12 35 48.—Pushing ether.

I. 12 37 0.—Pushing chloroform until death.

(The manometer tracing was again interrupted before the end of the experiment.)

### November 25th.—No. 117.

Temperature of the room 23 Cent. Unusually large brindled dog. Weight 38¾ lbs. Into the chloroform box at 7h. 28m. 10s. Placed on the table at 7h. 36m. 20s., and kept quiet with chloroform. Left carotid ligatured at 7h. 46m. 10s. Canula inserted at 7h. 49m. 15s. Temperature in rectum 101·4 F. Connection made with the manometers at 7h. 56m. 15s. Four Ludwig and six Fick tracings showing the effect of pushing chloroform during irritation of the entire right vagus on several occasions. In one of these the irritation of the vagus was kept up continuously for more than eight minutes, and the chloroform administration for seven minutes and the animal recovered without artificial respiration. At another time when the irritation accidentally failed on account of defect in the electrodes, the animal was with difficulty restored after chloroform had been pushed for one minute. Death eventually ensued after prolonged irritation of the vagus together with pushing chloroform and artificial respiration to ensure the chloroform entering the lungs freely.

*Observations.*

H. M. S.

A. 7 56 0.—Chloroform on an ordinary cap.

B. 8 1 40.—Electrical irritation of the right vagus. Inhibition of the heart is seen in Fick 4.

C. 8 2 50.—Chloroform pushed to stoppage of respiration ; artificial respiration ; irritation of right vagus during revival (*vide* Fick 5).

D. 8 9 10.—Irritation of right vagus, chloroform commenced at 8h. 9m. 20s. and pushed to stoppage of respiration (8h. 10m. 25s), chloroform stopped at 8h. 11m. 50s. ; irritation of the vagus stopped at 8h. 12m. 10s. Natural breathing at once recommenced spontaneously. Observation repeated in the same way and with the same result at 8h. 15m. 30s. (*vide* Fick 6.) Observation repeated for a much longer time at 8h. 28m. 30s. The chloroform and irritation of the vagus were kept up from 8h. 28m. 30s. to 8h. 36m. 40s. (*vide* Fick 9, 10, 11, 12, 13 and 14). Directly the irritation was stopped the pressure rose, showing that very little chloroform had hitherto been conveyed to the nerve centres, and that there is no danger in mere lowering of the blood-pressure. The pressure fell again at 8h. 37m. 0s. This evidently occurred from the inhibition of the heart having been

H. M. S.

stopped. The residual chloroform in the lungs was then rapidly taken up and produced its ordinary effect. (Compare with Observation D in Experiment 64).

E. 8 52 30.—Chloroform administration for one minute and twenty-five seconds. Respiration stopped at 8h. 53m. 55s. Artificial respiration was then performed for two and-a-half minutes, from 8h. 54m. 0s., and natural breathing recommenced at 8h. 57m. 0s.

F. 9 2 20.—Electrical irritation of vagus ; the stimulation was effective at first (*vide* Fick 19), but wore off and when chloroform was administered at 9h. 3m. 0s. it had almost ceased, and the full effects of the anæsthetic were quickly developed. Respiration stopped at 9h. 4m. 0s. and the chloroform cap was removed at 9h. 4m. 15s. Though the anæsthetic had only been administered for little over a minute, the animal was restored with great difficulty by artificial respiration, which was kept up for six minutes and thirty-five seconds before natural breathing returned at 9h. 11m. 0s.

G. 9 19 20.—Chloroform pushed again with irritation of the vagus at the same time. In this observation the stimulation did not become effective till after the respiration stopped. The breathing ceased at 9h. 20m. 20s. The irritation was stopped at 9h. 21m. 25s. and the chloroform cap was removed at 9h. 21m. 35s. Breathing recommenced spontaneously at 9h. 23m. 50s., but was not fully established till 9h. 25m. 15s. During this time there were some remarkable fluctuations of the blood-pressure, the falls being probably due to residual chloroform being taken up from the lungs, alternating with rises due to elimination of the anæsthetic from the lungs when the animal breathed (*vide* Ludwig III. at 9h. 22m. 0s., 9h. 24m. 0s. and 9h. 25m. 0s.)

H. M. S.

H. 9 31 25.—This observation consisted in prolonged irritation of the vagus from 9h. 30m. 25s. to 9h. 41m. 50s. and the administration of chloroform by artificial respiration from 9h. 31m. 45s. to 9h. 39m. 50s. The animal died, but the heart did not stop finally till 9h. 44m. 30s. This observation would have been more satisfactory if the vagus irritation had been kept up until all the residual chloroform in the lungs had been got rid of by artificial respiration.

The whole experiment is most instructive, as it, like Observation D in Experiment No. 64, proves that inhibition of the heart's action by the vagus prevents the fatal effects of chloroform poisoning and benefits the heart. The animal in Experiment 117 was put into a condition of extreme danger, from which it could only be restored by means of artificial respiration, by inhalation of chloroform for little over one minute (*vide* Ludwig I., 8h. 3m. 0s. and Ludwig II., 8h. 53m. 30s). The same animal recovered spontaneously and readily after five minutes of chloroform inhalation, with inhibition of the heart produced by electrical stimulation of the vagus. At 8h. 29m. 20s. (*vide* Ludwig II.) chloroform was pushed for seven minutes during continued irritation of the vagus, and the animal came round without artificial respiration. The danger really begins in these cases when the irritation is discontinued or fails to inhibit the heart, and thus enables the residual chloroform in the lungs to be rapidly absorbed and thrown into the system (*vide* Ludwig III., 9h. 22m. 0s. to 9h. 25m. 0s).

November 25th.—No. 118.

Monkey (Macacus). Weight 11 lbs. Temperature of the room 24·5 Cent. Into chloroform box at 10h. 27m. 27s. Fallen down at 10h. 32m. 25s. Placed on table at 10h. 32m. 50s. and kept quiet with chloroform. Temperature in rectum 104·4 F. at 10h. 42m. 30s. Artery ligatured at 10h. 50m. 50s. Canula inserted at 10h. 52m. 30s. Both vagi looped with a loose ligature. Connection made with manometer a few seconds before 10h. 58m. Two Ludwig and three Fick tracings.

*Observations.*

H. M. S.

A. 10 58 55.—Ordinary chloroform administration with violent struggling.

B. 11 3 20.—Irritation of the entire left vagus.

H. M. S.

C. 11 33 40.—Irritation of the left vagus and pushing chloroform at the same time on two occasions.

D. 11 41 40.—Chloroform administration with struggling.

E. 11 42 20.—Imperfect irritation of the right vagus and pushing chloroform.

The tracing was interrupted by clotting in the tubes just as the irritation of the vagus and chloroform administration were stopped. The animal breathed spontaneously for a few seconds, but then finally ceased breathing and died.

### November 25th.—No. 119.

Temperature of the room 25 Cent. Large pariah, weight 30 lbs., that has had phosphorus on the previous two days, one grain each day, and had been chloroformed in the morning by Dr. Rustomji, and is extremely feeble. Into chloroform box at about 2h. 19m. Fallen down at 2h. 21m. Placed on the table at 2h. 22m. 10s. and kept quiet with chloroform. Respiration very feeble and artificial respiration began at 2h. 24m. 45s. Breathing better at 2h. 25m. 40s. (The dog was so feeble generally that it was not necessary nor advisable to tie him down on the board in the usual way.) Temperature 99·8 F. in the rectum at 2h. 29m. Connection made with manometer at 2h. 41m. 20s. Four Ludwig and three Fick tracings.

### *Observations.*

H. M. S.

A. 2 42 40.—Chloroform administration; holding breath and gasping; placing thermometer in the rectum; struggling.

B. 3 12 55.—Irritation of right vagus and pushing chloroform until respiration stopped: with spontaneous recovery (*vide* Fick 5).

C. 3 23 0.—Irritation of right vagus and, pushing ether (*vide* Fick 8).

D. 3 38 20.—Pushing chloroform, and after the blood-pressure had fallen, irritating the vagus.

E. 3 43 20.—Artificial respiration.

F. 3 53 30.—Tracheotomy.

H. M. S.

G. 3 55 0.—Artificial respiration by bellows apparatus attached to a tube in the trachea.

H. 4 10 0.—Blowing ether by means of this apparatus directly into the trachea.

I. 4 13 40.—Introduction of chloroform by means of a Junker's inhaler introduced through the tracheal tube down to the bronchi, and pushing it until death resulted. Post-mortem.—Temperature 97·6 F. Heart pale and rather friable. Liver very pale and soft.

November 25th.—No. 120.

Rabbit that had had 5 minims of liquor atropiæ an hour before. Into chloroform box, 1 cubic foot in area, at 4h. 28m. 30s. Drowsy and vessels of the ear dilated at 4h. 29m. Fallen on its side and whining at 4h. 29m. 30s. Breathing stopped at 4h. 30m. Taken out at once and artificial respiration commenced, but this was of no avail though continued until 4h. 38m.

November 26th.—Nos. 121 and 122.

No. 121. Rabbit. 5 minims of solution of atropia sulphate injected into the thigh at 7h. 10m. 20s.

No. 122. Rabbit that has not had atropine :—

| | No. 121. | No. 122. |
|---|---|---|
| | H. M. S. | H. M. S. |
| Both into chloroform at | 7 25 15 | 7 25 15 |
| Fell down on side at | 7 27 5 | 7 27 5 |
| Sat up again at | 7 27 30 | Continued on his side |
| Struggling at | 7 27 40 | |
| Crying at | ... | 7 28 45 |
| Stertorous breathing at | ... | 7 29 7 |
| Fundus of the eye bluish at | ... | 7 29 30 |
| Beating time with feet at | 7 30 15 | 7 30 15 |
| Stopped breathing at | ... | 7 31 5 |
| Taken out of box and artificial respiration commenced at | ... | 7 31 30 |
| Breathing naturally at | ... | 7 32 30 |
| Cornea sensitive at | ... | 7 32 45 |
| Taken out of box though still breathing at | 7 33 10 | ... |
| Cornea sensitive at | 7 34 26 | ... |
| Excited struggling at | 7 35 0 | ... |

At 7h. 38m. the pupils of 122 were more dilated than the pupils of 121.

## November 26th.—Nos. 123 and 124.

| | No. 123. | | | No. 124. | | | |
|---|---|---|---|---|---|---|---|
| Two small monkeys, weight ... ... ... | 7 lbs. | | | 8¼ lbs. | | | |
| | H. | M. | S. | H. | M. | S. | |
| Into chloroform in a cubic foot box at ... ... ... | 7 | 53 | 0 | 7 | 53 | 0 | |
| Fallen down at ... ... ... ... ... ... | 7 | 56 | 30 | 7 | 56 | 30 | |
| Struggling at ... ... ... ... ... ... | | ... | | 7 | 56 | 40 | |
| Taken out of box at ... ... ... ... ... | 7 | 57 | 40 | 7 | 57 | 40 | |
| Chloroform to keep quiet at ... ... ... ... | 7 | 59 | 40 | 7 | 59 | 40 | |
| "Under." Stopped chloroform at ... ... ... | 8 | 0 | 30 | 8 | 0 | 45 | |
| More chloroform again at ... ... ... ... | 8 | 1 | 53 | 8 | 6 | 32 | |
| "Under." Stopped chloroform at ... ... ... | 8 | 2 | 30 | 8 | 7 | 50 | |
| More chloroform 3rd time on inhaler in front of tracheal tube at ... ... ... ... ... ... ... | 8 | 7 | 50 | 8 | 10 | 30 | |
| "Under." Stopped chloroform at ... ... ... | 8 | 7 | 53 | 8 | 11 | 52 | |
| More chloroform, 4th time at ... ... ... ... | 8 | 11 | 10 | 8 | 21 | 30 | Do. |
| "Under." Chloroform stopped at ... ... ... | 8 | 11 | 50 | 8 | 23 | 10 | |
| More chloroform, 5th time at ... ... ... ... | 8 | 16 | 50 | | ... | | |
| "Under" at ... ... ... ... ... ... | 8 | 18 | 0 | | ... | | |
| Opened trachea at... ... ... ... ... ... | 8 | 9 | 0 | 8 | 7 | 35 | |
| Canula inserted at... ... ... ... ... ... | 8 | 9 | 5 | 8 | 8 | 45 | |
| Re-inserted, having slipped out at ... ... ... | 8 | 20 | 7 | | ... | | |
| Attached to the bellows artificial respiration apparatus, which was so arranged as to blow air through a separate bottle into each monkey at ... ... ... | 8 | 23 | 27 | 8 | 23 | 27 | |
| Opening the thorax at ... ... ... ... ... ... | 8 | 24 | 30 | 8 | 24 | 0 | |
| Heart exposed at ... ... ... ... ... ... | 8 | 26 | 30 | 8 | 27 | 0 | |
| Ether in bottle at... ... ... ... ... ... | | ... | | 8 | 32 | 15 | |
| Chloroform in bottle at ... ... ... ... ... | 8 | 32 | 30 | | ... | | |
| Heart stopped ... ... ... ... ... ... | 8 | 39 | 0 | | ...* | | |

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* Owing to a kink in the tubes No. 124 was killed by asphyxia; his heart only flickering or very feebly contracting when it was first exposed, but it continued to do so until 10h. 48m. with still a faint contraction of one corner of the auricle at 11h. 30m.

## November 26th.—No. 125.

Monkey (medium-sized Macacus). Weight 11¾ lbs. Into chloroform box at 9h. 20m. Fallen down at 9h. 29m. 15s. Placed on board at 9h. 29m. 30s. Kept quiet with chloroform. Trachea opened at 9h. 35m. 30s. Tube inserted at 9h. 36m. Artificial respiration kept up by bellows apparatus and chest opened. Attempt to connect with Roy's cardiomyograph failed. Ether began at 10h. 22m. 10s. Heart contracting very feebly. Fresh air 10h. 24m. 30s. Heart just moving and no more. Flickering until 10h. 40m.

### November 26th—No. 126.

Monkey, small. Weight 4½ lbs. Into box and chloroform given at 10h. 36m. 30s. Fallen down at 10h. 40m. 45s. Trachea opened at 10h. 45m. 5s. and canula inserted. Kept quiet with chloroform. Opening thorax at 11h. 6m. 15s. with artificial respiration by bellows apparatus. Heart exposed at 11h. 8m. 30s. 5 cc. of ether into the bottle of the apparatus. Animal "quite out" at 11h. 11m. Cornea insensitive at 11h. 11m. 50s. Heart's action very rapid at 11h. 13m. 50s., but still acting vigorously. Chloroform, same quantity, at 11h. 16m. 45s. (all the ether had evaporated). Ventricle stopped at 11h. 17m. 45s. Heart flickering at 11h. 19m. Lungs no longer contracting at 11h. 20m. 30s. All right again at 11h. 21m. 50s. Lungs not distending at 11h. 24m. owing to apparatus breaking down. 3¼ cc. of chloroform remained in the bottle.

| November 26th—No. 127.<br>Small monkey. Weight 4½ lbs. | H. | M. | S. | November 26.—No. 128.<br>Small monkey. Weight 4¼ lbs. |
|---|---|---|---|---|
| Into chloroform box 1 cubic foot capacity. | 2 | 32 | 0 | Into chloroform box 1 cubic foot capacity. |
| Still standing and licking the glass ... | 2 | 33 | 45 | Fallen down. |
| More chloroform into the box ... ... | 2 | 35 | 45 | Taken out of box and tied on to board. |
| Fallen down ... ... ... ... | 2 | 37 | 0 | |
| Taken out of box and tied on board ... | 2 | 37 | 10 | |
| | 2 | 38 | 0 | Tracheotomy commenced. |
| More chloroform in ordinary cap ... ... | 2 | 39 | 41 | |
| Stopped getting chloroform ... ... | 2 | 40 | 10 | |
| | 2 | 40 | 20 | Trachea opened. |
| Tracheotomy commenced ... ... ... | 2 | 40 | 35 | |
| | 2 | 41 | 5 | Glass tube inserted. |
| | 2 | 41 | 53 | Having chloroform (cap held before tube). |
| | 2 | 43 | 3 | Stopped chloroform. |
| More chloroform ... ... ... ... | 2 | 43 | 37 | |
| Stopped chloroform ... ... ... | 2 | 43 | 58 | |
| More chloroform ... ... ... ... | 2 | 45 | 38 | |
| Stopped again ... ... ... ... | 2 | 46 | 5 | |
| Trachea opened ... ... ... ... | 2 | 47 | 30 | |
| | 2 | 49 | 17 | More chloroform. |
| Glass tube into trachea ... ... ... | 2 | 50 | 27 | |
| | 2 | 51 | 4 | Stopped chloroform. |
| More chloroform on ordinary cap in front of tube. | 2 | 51 | 35 | |
| Stopped chloroform... ... ... ... | 2 | 52 | 43 | |

| No. 127—*continued.* | H. | M. | S. | No. 128—*continued.* |
|---|---|---|---|---|
| Tube slipped out of trachea | 2 | 53 | 10 | |
| | 2 | 53 | 45 | More chloroform. |
| | 2 | 54 | 15 | Stopped chloroform. |
| More chloroform by mouth | 2 | 54 | 40 | |
| Stopped chloroform | 2 | 55 | 10 | |
| Glass tube inserted again | 2 | 55 | 31 | |
| More chloroform | 2 | 55 | 52 | |
| Stopped chloroform | 2 | 56 | 28 | |
| | 2 | 57 | 43 | More chloroform. |
| More chloroform | 2 | 58 | 54 | |
| Stopped chloroform | 2 | 59 | 30 | Stopped chloroform. |
| Connected with the bellows-artificial respiration apparatus. | 3 | 0 | 0 | Connected with the bellows-artificial respiration apparatus. |
| Opening thorax | 3 | 0 | 21 | Opening thorax. |
| Chloroform through the bellows | 3 | 0 | 44 | Chloroform through the bellows. |
| | 3 | 1 | 52 | Heart exposed. |
| Heart exposed | 3 | 2 | 4 | |
| Struggling | 3 | 2 | 25 | Struggling. |
| | 3 | 3 | 10 | Lungs not expanding. |
| | 3 | 3 | 20 | Expanding imperfectly. |
| Lungs expanding freely | 3 | 4 | 50 | Lungs expanding freely. |
| | 3 | 5 | 10 | Ether into the bottle. |
| Chloroform into the bottle | 3 | 5 | 30 | |
| Heart distinctly feeble | 3 | 5 | 25 | |
| | 3 | 5 | 38 | Heart very rapid. |
| | 3 | 7 | 7 | Jerky action of the diaphragm. |
| Needle into heart | 3 | 7 | 20 | Needle into heart. |
| Another needle into heart | 3 | 7 | 30 | |
| | 3 | 8 | 45 | More ether into bottle. Cornea sensitive. |
| Lungs not expanding | 3 | 9 | 40 | |
| Heart stopped | 3 | 12 | 5 | |
| (Post-mortem showed clot at the bifurcation of the bronchi). | 3 | 14 | 45 | Ether completely evaporated. |
| | 3 | 15 | 5 | More ether into bottle. |
| | 3 | 17 | 30 | Needle barely moving. |
| | 3 | 27 | 45 | Stopped. |

## November 26th.—No. 129.

## November 26th.—No. 130.

| Small monkey. Weight 3¾ lbs. | H. | M. | S. | Small monkey. Weight 4¼ lbs. |
|---|---|---|---|---|
| Into chloroform box | 3 | 31 | 32 | Into chloroform box. |
| Drunk | 3 | 34 | 52 | Drunk. |
| Fallen down | 3 | 34 | 10 | |

| No. 129—*continued.* | H. | M. | S. | No. 130—*continued.* |
|---|---|---|---|---|
| | 3 | 34 | 22 | Fallen down. |
| Taken out of box | 3 | 35 | 7 | Taken out of box. |
| | 3 | 35 | 35 | More chloroform. |
| More chloroform | 3 | 36 | 0 | Stopped chloroform. |
| Stopped chloroform | 3 | 36 | 32 | |
| | 3 | 36 | 57 | More chloroform. |
| More chloroform | 3 | 37 | 24 | |
| | 3 | 37 | 58 | Stopped chloroform. |
| Commenced tracheotomy | 3 | 38 | 40 | |
| | 3 | 38 | 50 | Commenced tracheotomy. |
| Stopped chloroform | 3 | 39 | 0 | |
| Trachea opened | 3 | 39 | 48 | |
| Glass tube inserted | 3 | 40 | 8 | |
| | 3 | 40 | 10 | Stopped chloroform (commencement not noted). |
| More chloroform | 3 | 40 | 45 | |
| Stopped chloroform | 3 | 42 | 0 | |
| More chloroform | 3 | 43 | 45 | |
| Stopped chloroform | 3 | 44 | 47 | |
| More chloroform | 3 | 46 | 30 | |
| | 3 | 46 | 50 | Tube inserted into trachea. |
| Stopped chloroform | 3 | 47 | 25 | |
| | 3 | 51 | 0 | More chloroform. |
| More chloroform | 3 | 51 | 52 | |
| Stopped chloroform | 3 | 52 | 27 | |
| More chloroform | 3 | 53 | 15 | |
| Stopped chloroform | 3 | 53 | 41 | Stopped chloroform. |
| Connected with artificial respiration apparatus. | 3 | 55 | 0 | |
| Cornea sensitive | 3 | 55 | 15 | Connected with apparatus; cornea sensitive. |
| Chloroform through bellows | 3 | 56 | 0 | Chloroform through bellows. |
| 10 minims chloroform into bottle | 3 | 57 | 25 | |
| | 3 | 57 | 43 | 10 minims chloroform into bottle. |
| Cornea insensitive | 3 | 58 | 0 | Cornea sensitive. |
| Opening chest | 3 | 58 | 15 | Opening chest. |
| Heart fully exposed | 3 | 59 | 10 | |
| Lungs expanding better | 4 | 0 | 0 | |
| | 4 | 0 | 20 | Heart exposed. |
| 5 cc. ether into bottle | 4 | 2 | 24 | |
| | 4 | 2 | 37 | 5 cc. chloroform into bottle. |
| Needle into heart | 4 | 2 | 57 | |
| Heart beating rapidly, communicating movements to the abdominal wall. | 4 | 3 | 30 | Heart very feebly beating. |

| No. 129—*continued.* | H. | M. | S. | No. 130—*continued.* |
|---|---|---|---|---|
| | 4 | 3 | 55 | Needle into heart. |
| 5 cc. more ether ... ... ... ... | 4 | 8 | 0 | |
| | 4 | 8 | 18 | 5 cc. more chloroform. |
| Needle moving less vigorously but distinctly. | 4 | 8 | 40 | Needle barely moving. |
| | 4 | 10 | 0 | Very slightly moving, if at all. |
| | 4 | 10 | 55 | Absolutely not moving. |
| Needle moving slowly but steadily. More ether. | 4 | 12 | 3 | After opening pericardium heart again flickered. |
| Heart stopped ... ... ... ... | 4 | 12 | 20 | |

## November 27th.—No. 132, 3½ lbs. — No. 131, 3½ lbs.

### Medium-sized monkey. — Medium-sized monkey.

| No. 132 | H. | M. | S. | No. 131 |
|---|---|---|---|---|
| Into chloroform box ... ... ... | 4 | 24 | 20 | Into chloroform box. |
| | 8 | 26 | 0 | Drooping. |
| More chloroform into box ... ... ... | 8 | 27 | 18 | More chloroform into box. |
| | 8 | 27 | 47 | Fallen against the side. |
| | 8 | 28 | 50 | Down and breathing stertorously. |
| | 8 | 29 | 55 | Taken out of box and tied on to a board. |
| Fallen down... ... ... ... ... | 8 | 30 | 10 | |
| Taken out and tied on to a board... ... | 8 | 31 | 8 | |
| | 8 | 31 | 32 | More chloroform into the box. |
| | 8 | 32 | 22 | Stopped chloroform and commenced tracheotomy. |
| More chloroform ... ... ... ... | 8 | 33 | 20 | |
| Stopped chloroform and begin tracheotomy. | 8 | 34 | 4 | |
| | 8 | 34 | 49 | Trachea opened. |
| | 8 | 35 | 9 | Tube inserted into trachea. |
| | 8 | 35 | 51 | More chloroform. |
| Trachea opened ... ... ... ... | 8 | 36 | 33 | |
| Tube inserted ... ... ... ... | 8 | 36 | 55 | |
| Respiration stopped & artificial respiration.. | 8 | 37 | 45 | |
| | 8 | 38 | 5 | Stopped chloroform. |
| | 8 | 39 | 15 | More chloroform. |
| | 8 | 40 | 2 | Stopped chloroform. |
| Pressure had probably been made on the chest by the operator, who had operated from below and covered the chest with his hands. | 8 | 43 | 20 | |
| No heart sound audible ; needle into heart.. | 8 | 44 | 15 | |
| Needle feebly moving ; continue artificial respiration. | 8 | 45 | 10 | More chloroform ; struggling. |
| Abandoned ... ... ... ... ... | 8 | 46 | 23 | Stopped chloroform. |

## No. 134.

## No. 131—*continued.*

Another medium-sized monkey.
Weight 4 lbs.

| No. 134 | H. | M. | S. | No. 131—*continued.* |
|---|---|---|---|---|
| Into chloroform box | 8 | 51 | 3 | |
| Still sitting up and looking about him | 8 | 52 | 46 | More chloroform. |
| Leaning against the side | 8 | 53 | 37 | Stopped chloroform. |
| Still moving about | 8 | 56 | 23 | |
| Fallen down | 8 | 57 | 58 | |
| Taken out of box | 8 | 58 | 5 | |
| More chloroform on cap | 8 | 59 | 0 | |
| Chloroform stopped ; tracheotomy begun | 9 | 1 | 25 | More chloroform. |
| | 9 | 1 | 52 | Stopped chloroform. |
| Trachea opened | 9 | 2 | 7 | |
| Tube inserted | 9 | 2 | 18 | |
| More chloroform | 9 | 3 | 9 | |
| | 9 | 3 | 29 | More chloroform. |
| | 9 | 3 | 50 | Stopped chloroform. |
| Stopped chloroform | 9 | 3 | 55 | |
| Attached to bellows apparatus | 9 | 4 | 5 | Attached to bellows apparatus. |
| | 9 | 4 | 31 | Opening thorax. |
| Commenced to open thorax and artificial respiration begun. | 9 | 5 | 5 | Artificial respiration begun. |
| A little chloroform into bottle | 9 | 6 | 45 | |
| | 9 | 6 | 27 | Heart exposed. |
| More chloroform into bottle | 9 | 7 | 27 | |
| " Over " and proceed with operation | 9 | 8 | 0 | |
| Heart exposed | 9 | 8 | 57 | |
| Pericardium opened | 9 | 9 | 40 | Chloroform into bottle. |
| | 9 | 10 | 30 | Quite over. |
| | 9 | 10 | 35 | Needle into heart. |
| | 9 | 11 | 20 | Bottle emptied. |
| | 9 | 11 | 35 | Needle taken out of heart. |
| Cornea sensitive | 9 | 12 | 30 | |
| | 9 | 12 | 50 | Ether into bottle 5 cc. |
| 5 cc. chloroform into bottle | 9 | 13 | 10 | |
| Needle into heart | 9 | 13 | 30 | Needle into heart. |
| | 9 | 15 | 5 | 5 cc. more ether into bottle. |
| 5 cc. chloroform | 9 | 15 | 45 | |
| | 9 | 17 | 0 | Heart beating rapidly. Ether all evaporated. |
| | 9 | 17 | 55 | 10 cc. more ether into bottle. |
| 5 cc. chloroform | 9 | 19 | 20 | |
| Left ventricle hardly acting ; right better | 9 | 21 | 20 | |
| | 9 | 21 | 55 | Pericardium removed. |

| No. 134—*continued.* | H. | M. | S. | No. 131—*continued.* |
|---|---|---|---|---|
| Heart feebly flickering . ... | 9 | 22 | 40 | |
| | 9 | 24 | 34 | 8 cc. ether into bottle. |
| | 9 | 29 | 43 | Another needle into heart. |
| Needle absolutely still . ... | 9 | 38 | 0 | |
| | 9 | 39 | 55 | Connection between needle and a time-marker (*vide* tracing). |
| | 9 | 44 | 15 | More ether into bottle. |
| | 9 | 51 | 5 | Artificial respiration stopped (after which it made spontaneous respiratory movements about every fourth second). |
| | 10 | 0 | 0 | Artificial respiration commenced. He came round and breathed spontaneously again (*vide* tracing II). |
| | 10 | 24 | 0 | Artificial respiration begun again. |
| | 10 | 27 | 0 | Chloroform into bottle. |
| | 10 | 37 | 0 | Needle taken out. Heart still beating. |
| | 10 | 48 | 30 | Finally stopped, but still remained irritable, so that it began beating again when needle was put into it. |

## November 27th.—No. 133.

Horse thrown and given chloroform on a nose-bag at 10h. 50m. Loop under right vagus at 10h. 55m. 45s. Right carotid ligatured at 10h. 57m. 40s. Canula inserted at 11h. 1m. Connection with manometer at 11h. 17m. 55s. while horse was on the floor. (He was afterwards, 11h. 42m. 30s., raised on to the table.)

Two Ludwig and two Fick tracings.

### *Observations.*

A. Ordinary chloroform administration.

B. Slight struggling.

C. Pushing chloroform until respiration stopped; death resulted in spite of long-continued artificial respiration.

Repeated attempts were made to irritate the vagus, but they failed as the nerve was never exposed.

## November 27th.—No. 135.

Medium-sized monkey. Weight 13 lbs. Temperature of room 23½ Cent. Into chloroform box at 2h. 22m. 35s. Placed on the table at 2h. 27m. 42s. and kept quiet with chloroform. Temperature in rectum 102·6 F. at 2h. 37m.

30s. Artery ligatured at 2h. 45m. 25s. Canula inserted at 2h. 48m. 10s. Loop under both vagi. Connection made with manometer at 2h. 59m. 50s. Three Ludwig and three Fick tracings.

*Observations.*

A. Ordinary chloroform administration and struggling.

B. Double ligature and division of the right vagus.

C. Irritation of the central, and

D. Peripheral end of the vagus.

E. Injection of 10 grains of chloral into the peritoneum, and afterwards irritation of the central and peripheral end of the right vagus.

F. Exposure and ligature of the femoral artery.

G. Opening the trachea and tying a small glass tube into it.

H. Closure of the tracheal tube so as to produce almost complete asphyxia.

I. Pumping chloroform vapour into the closed tube by means of Junker's inhaler while the respirations are still embarrassed and consequent speedy death.

While the chest was being opened, air suddenly rushed in, and it was thought that spontaneous respiration was going to recommence, but there was no real spontaneous movement. After death chloroform was injected with much force into the femoral artery and produced complete rigidity of the limb.

November 25th.—No. 136.

Goat, young male. Weight 16 lbs. Into chloroform box at 10h. 48m. 20s. Fallen down and taken out of box at 10h. 59m. 20s. Placed on the board and kept quiet with chloroform. Loop under both vagi. Ligatured the left carotid at 11h. 9m. 26s. Canula inserted at 11h. 11m. 47s. Temperature in rectum 102·6 F. at 11h. 18m. 20s. Connection with manometers at 11h. 19m. 20s. One Ludwig and one Fick tracing.

H. M. S. *Observations.*

A. 11 11 30.—Ordinary chloroform administration ; holding breath.

B. 11 25 10.—Pushing chloroform until respiration had almost ceased.

C. 11 30 0.—General convulsions ; respiration ceased at 11h. 28m. 20s.

H. M. S.

D. 11 30 30.—Artificial respiration was commenced some minutes after the respiration had entirely ceased and failed to restore the animal. The case was complicated by a very distended stomach and the fact that the trachea was filled with fluid at an early stage. It was thought that some of the contents of the stomach found their way into the air passages during the convulsions or even before they occurred. A needle was inserted into the heart at 11h. 34m. 0s. and moved rhythmically until 11h. 41m. 40s.

November 28th.—No. 137.

Goat, young male, weight 15¾ lbs. Into chloroform box at 11h. 46m. 43s. Fell down at 12h. 2m. 30s. and taken out of box, but still quite sensitive. More chloroform from time to time to keep it under. Holds its breath like the other goat whenever chloroform is given. Temperature in rectum at 12h. 8m., 103·6 F. Artery ligatured at 12h. 11m. 15s. Canula inserted at 12h. 12m. 50s. Connection with manometer at 12h. 16m. Two Ludwig and one Fick tracing.

*Observations.*

H. M. S.

A. 12 18 10.—Ordinary chloroform administration showing the effect on the tracing of holding the breath, and of irregular jerking respiration.

B. 12 25 40.—Irritation of the left vagus nerve (*vide* Fick 4).

C. 12 37 30.—Pushing chloroform and irritating the left vagus (*vide* Fick 6).

D. 12 45 40.—The same, only for a much longer time, after which the animal died in spite of artificial respiration, which however, for some reason, was never efficient, no air passing in and out of the chest.

On opening the chest a large quantity of blood was found in the left pleural cavity.

November 29th.—No. 138.

Temperature of room 20 Cent. Cat, weight 6½ lbs. Into chloroform at 7h. 58m. 5s. More chloroform into the box at 8h. 1m. 55s. Struggling at 8h. 2m. Fallen down at 8h. 2m. 15s. Tied on to rabbit board and given chloroform from time to time to keep it under. Temperature in the rectum 100·6 F. at 8h. 9m. 45s. and artery ligatured. Connection with manometer at 8h. 1m. 25s.

Two Ludwig and one Fick tracing.

*Observations.*

H. M. S.
A. 8 4 30.—Administration of ether on a cap.
B. 8 34 0.—Struggling.
C. 8 44 30.—Pushing ether until respiration had almost ceased and only occasional gasping continued.
D. 8 53 30.—Pushing ether with the cap covered with mackintosh until death ensued.

In this and the next experiment the respiration was registered by a system of Marey's tambours connected with a pin in the chest wall, the marker running below the manometer tracing.

November 29th.—No. 139.

Small cat, weight 6¼ lbs. Chloroform given at 9h. 29m. 20s. Fallen down at 9h. 31m. 45s. Placed on the table at 9h. 35m. 40s. During the operation of exposing the carotid the cornea was quite insensitive, but there was whining respiration. Artery ligatured at 9h. 40m. 50s. Canula inserted at 9h. 42m. 20s. Connection with manometer at about 9h. 50m. 30s. One Ludwig and one Fick tracing.

*Observations.*

H. M. S.
A. 9 51 50.—Ordinary chloroform administration.
B. 9 58 50.—Pushing chloroform until death ensued.

The trace was interrupted just before the end by clots in the tube.

November 29th.—No. 140.

Moderate-sized monkey, weight 11¼ lbs. Temperature of the room 21 Cent. Into chloroform in a cubic foot box at 10h. 42m. 15s. Fallen against the side of the box at 10h. 47m. 20s. A little more chloroform into box at 10h. 48m. 45s. Down at 10h. 49m. 10s. Eyes still blinking. Taken out of box at 10h. 50m. 20s. and kept quiet with chloroform. Temperature 103 F. in rectum. Canula inserted into carotid 11h. 5m. 30s.

One Ludwig and one Fick tracing.

*Observations.*

H. M. S.
A. 11 12 40.—Ether administration on a cap covered with a small piece of mackintosh which failed to keep the animal thoroughly under, though the cornea was insensitive, struggling occurring when a pin was thrust into the chest.

B. 11 20 35.—Ether administration *plus* asphyxia by enveloping the head completely in mackintosh.

C. 11 27 50.—Asphyxia by the same means without ether.

D. 11 32 20.—Pushing ether *plus* asphyxia until death resulted.

The trace was interrupted before the death of the animal by a clot in the tubes.

November 29th.—No. 141.

Large goat, weight 66 lbs. Chloroformed by a nose-bag while standing at 2h. 18m. 51s. Thrown down at 2h. 19m. 50s. Cornea insensitive at 2h. 20m. 40s. and chloroform stopped, the animal lying quite quiet while being tied down on to the table. Some salivation. Cornea sensitive at 2h. 22m. 40s. More chloroform at 2h. 23m. 10s. Cornea insensitive at 2h. 25m. 30s. and chloroform stopped. Left carotid ligatured at 2h. 27m. 40s. More chloroform at 2h. 31m. 28s. Stopped chloroform at 2h. 33m. 3s. More chloroform at 2h. 35m. 48s. Stopped chloroform at 2h. 36m. 35s. More chloroform at 2h. 38m. 50s. Canula inserted into the artery at 2h. 41m. Stopped chloroform at 2h. 41m. 52s. More chloroform at 2h. 43m. 31s. Connection made with manometers at 2h. 44m. 45s.

One Ludwig and one Fick tracing.

*Observations.*

H. M. S.
A. 2 48 40.—Pushing chloroform until respiration ceased (*vide* Fick 3).

B. 2 52 0.—Artificial respiration.

C. 2 58 35.—Pushing chloroform until death ensued.

D. 3 1 0.—Thrusting needle into the heart. The depressing effects of this operation is seen in the Ludwig trace.

Post-mortem.—Temperature 99·6 F.

November 20th.—No. 142.

Horse thrown and given chloroform at 3h. 19m. 45s. very freely,* and while struggling chloroform had been stopped about one minute (?) when respiration was noticed to have stopped at 3h. 23m. 30s. Artificial respiration commenced at once. No pulse at 3h. 25m. 45s., and the animal was not restored to life.

November 29th.—No. 143.

(*In the presence of His Highness the Nizam.*)

Large goat, weight 70 lbs., given chloroform in a bag while standing at 3h. 35m. 55s. Thrown down at once. Still bleating at 3h. 37m. 5s. Stopped chloroform at 3h. 37m. 50s., and while he was being carried and put on the table respiration stopped at 3h. 38m. Artificial respiration commenced at once ; breathing naturally 3h. 38m. 55s. Cornea sensitive. More chloroform at 3h. 42m. 25s. Stopped at 3h. 43m. 2s. Artery ligatured at 3h. 43m. 20s. Temperature 102 F. in rectum. Temperature of the room 23½ Cent. Canula inserted at 3h. 46m. 15s. More chloroform at 3h. 47m. 30s. Connection made with the manometers at 3h. 50m. 30s. One Ludwig and one Fick tracing.

*Observations.*

H. M. S.

A. 3 51 0.—Ordinary chloroform administration.

B. 3 51 30.—Violent struggling (during which the marker was pushed over the top of the drum on several occasions by the violent oscillations in the pressure, and the tracings are consequently imperfect).

C. 3 58 0.—Pushing chloroform until respiration ceased.

D. 4 3 50.—Artificial respiration.

E. 4 5 10.—Pushing chloroform until respiration had ceased for some time, and failure to restore the animal by artificial respiration.

F. 4 9 30.—The last ineffective gasps are shown on the Ludwig tracing.

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* A pint of chloroform was said to have been poured into the nose-bag.

### November 29th.—No. 144.

*(In the presence of His Highness the Nizam.)*

Small horse, thrown at 4h. 24m. 15s. Given chloroform in a nose-bag at 4h. 26m. 55s. Struggling until 4h. 28m. 35s. Operation to tie artery commenced at 4h. 28m. 55s. Stopped chloroform at 4h. 29m. 35s. More chloroform at 4h. 30m. 10s. Stopped chloroform at 4h. 31m. 15s. Loop under left vagus. Ligature left carotid artery at 4h. 31m. 50s. More chloroform at 4h. 33m. 17s. Stopped chloroform at 4h. 35m. 40s. Canula inserted at 4h. 36m. More chloroform at 4h. 37m. 30s. Stopped chloroform at 4h. 39m. One Ludwig and one Fick tracing.

*Observations.*

H. M. S.

A. 4 44 0.—Ordinary chloroform administration.

B. 4 47 10.—Irritation of left vagus three times, the second and third noticed on Fick readings 2 and 3.

C. 4 50 30.—Pushing chloroform until respiration ceased.

D. 5 0 0.—Artificial respiration. Pushing chloroform for 9 minutes with, at the same time, irritation of the left vagus for more than 7 minutes.

The respiration ceased after the irritation was discontinued and the chloroform was then stopped. Pressure continued to fall and artificial respiration failed to restore the animal. The trace ends after 5h. 11m., but artificial respiration was continued until 5h. 15m. A needle then thrust into the heart did not move. Thorax opened and heart still at 5h. 20m.

### November 29th.—No. 145.

*(In the presence of His Highness the Nizam.)*

Monkey, large-sized female (Macacus). Into chloroform box, one cubic foot, at 5h. 33m. 20s. Fallen against the side at 5h. 34m. 30s. Fallen and taken out of box at 5h. 35m. 27s. More chloroform at 5h. 36m. 14s. Stopped chloroform at 5h. 37m. 5s. Commenced incisions in the neck 5h. 37m. 43s. More chloroform at 5h. 39m. 20s. Stopped chloroform 5h. 40m. 5s. Divide sterno cleido mastoid muscle at 5h. 40m. 27s. Loop under left vagus at 5h. 41m. 37s. More chloroform at 5h. 42m. 6s. Stopped chloroform at 5h. 42m. 43s. Open trachea at 5h. 42m. 47s. Tube inserted into trachea at 5h. 43m. Opening chest wall while artificial respiration maintained by means of a Junker bellows. Chloroform from time to time a few whiffs at 5h. 44m. 15s. Opening the pericardium at 5h. 47m. 50s. Needle into heart at 5h. 48m. 30s. Irritation left vagus, coil 5, at 5h. 50m. 35s. Stopped irritation at 5h. 50m. 47s. Irritation left vagus at 5h. 51m. 10s. Stopped irritation at 5h. 51m. 27s. Chloroform at 5h. 52m. 3s. Irritation of left vagus at 5h. 52m. 37s. Stopped irritation at 5h. 52m. 47s. Stopped artificial respiration and chloroform 5h. 53m. 22s. Diaphragm contracting rhythmically. Heart slow and feeble at 5h. 53m. 30s. Heart almost stopped. Began artificial respiration again at 5h. 54m. 35s. Heart more vigorous at 5h. 55m. 27s. Irritation of left vagus not acting made him cough, coil 6, at 5h. 56m. Irritation left vagus, coil 6, at 5h. 56m. 35s. acting fully. Irritation of left vagus continued at 5h. 56m. 57s. Chloroform at 5h. 57m. 15s. Stopped irritation at 5h. 57m. 40s. Irritation of left vagus again at 5h. 58m. 23s. Coils run up to zero at 5h. 59m. 20s. The heart stopped at first, but is now going again. Stopped irritation at 5h. 59m. 46s. Irritation of vagus again at 6h. 0m. 20s. Run up to zero at 6h. 0m. 30s. Stop up outlet for air at 6h. 2m. 23s. Stop chloroform and artificial respiration at 6h. 2m. 55s. Stop irritation at 6h. 3m. 10s. Air let out of the chest at 6h. 3m. 35s. Artificial respiration again at 6h. 3m. 55s. Heart hardly beating at 6h. 5m. 15s. Heart stopped at 6h. 6m. 30s.

### November 30.—No. 146.

Dog, weight 35 lbs., that has had three doses of phosphorus, one grain each day, on the 25th, 26th, and 27th instant, respectively. [Of twelve dogs similarly dosed four have already died.] Into chloroform box at 10h. 27m. 50s. Fallen down at 10h. 32m. 25s. On to the table at 10h. 33m. 15s. and given chloroform from time to time. Left carotid ligatured and canula inserted into it. Connection with manometers at 10h. 56m. 35s. Two Ludwig and one Fick tracing during administration of ether persistently, with more or less perfect exclusion of air, until death resulted. Both sides of the heart distended with venous blood. Heart and liver both fatty.

*Observation.*

H. M.

A. 10 50 0.—Ether administered with very little air. This observation, especially the Fick readings, shows again that ether has precisely the same action as chloroform, but is less intense.

## November 30th.—No. 147.

Another pariah to which phosphorus has been given as in the case of 146. Is, however, more sickly. Into chloroform box at 11h. 32m. 57s. Fallen down at 11h. 40m. 41s. Placed on the table and kept quiet with chloroform. Canula inserted into artery at 11h. 51m. 7s. One Ludwig and one Fick tracing during the administration of chloroform persistently until death resulted. At the commencement there was a sudden fall of pressure—the result of holding the breath.

### *Observation.*

H. M. S.

A. 11 59 45.—Chloroform. The animal held its breath, the effect being well seen in Fick 3. The pressure fell rapidly and several deep gasps were taken. The respiration stopped at 12h. 1m. 25s. The heart continued beating till 12h. 8m. 0s.

## November 30th.—No. 148.

A thin phosphorus dog of the same batch as 146 and 147. (Has had chloroform this morning, but was revived by artificial respiration.) Into chloroform box at 2h. 30m. 37s. Fallen down at 2h. 40m., and taken out of box. Placed on the table and kept under with chloroform. Artificial respiration at 2h. 42m. 20s. until 2h. 42m. 50s. Artery ligatured and canula inserted. Connections with manometers at 2h. 49m. 50s. Three Ludwig and four Fick tracings showing the effect of (*a*) giving chloroform on a cap crammed on closely over the face so as to partially asphyxiate the animal, compared with the gradual fall of pressure that occurs when chloroform is properly administered with air; (*b*) giving ether in the same way; (*c*) giving ammonia in the same way; (*d*) holding the dog's mouth and nose so as to produce asphyxia without chloroform or ether, and (*e*) irritation of the right vagus. The dog was eventually killed by making him inhale concentrated chloroform vapour through a tube tied into the trachea.

### *Observations.*

H. M. S.

A. 2 50 0.—Chloroform on a cap held close over the face; the animal struggled and held its breath. There was a rapid and irregular fall of pressure (*vide* Fick 2).

B. 2 53 30.—Chloroform in the same way; same effects produced (*vide* Fick 3).

H. M. S.

C. 3 1 30.—Chloroform in the same way; same effects produced, but to such a marked degree that it was thought a clot had collected in the tubes (*see* Fick 4). Interruption.

D. 3 11 0.—Chloroform as before; same effects produced (note Fick 6).

F. 3 17 0.—Chloroform with sufficient air to ensure regular breathing; there was no struggling or holding of the breath, and no irregularity of the blood-pressure or circulation. Fick 7 is worthy of notice, though it records the pulse as the animal was almost coming round.

G. 3 21 10.—Chloroform with insufficient air; same effects as at A, B, & C; shown very markedly in Fick 8.

H. 3 35 0.—Inhalation of ammonia.

I. 3 36 45.—Chloroform again with plenty of air. There was slight struggling at first and slight corresponding irregularity in the fall of blood-pressure. Fick 12, taken when the pressure was getting very low, shows beautifully the regularity of the pressure and pulse tracing when the breathing is regular, and ought to be contrasted and compared with Fick 2, 3, 4, 6, 8, and 11.

J. 3 47 50.—Ether on cap held close over the face; struggling and holding the breath. Fick 14 and 15 show effects on the pressure and pulse, similar to those produced when chloroform is given in the same way. Compare Fick 14, 15, and 16 with Fick 7, 8, and 9.

K. 3 54 20.—Simple asphyxia showing rapid fall of pressure and irregular pulse (*vide* Fick 17).

L. 4 1 25.—Chloroform again with plenty of air. Breathing slightly irregular from groaning; there is a corresponding slight irregularity in the tracing.

M. 4 15 15.—Electrical irritation of right vagus: coil 10.

N. 4 16 40.—Chloroform again with sufficient air to ensure regular breathing. This observation was interrupted by clotting in the tubes.

H. M. S.

O. 4 20 0.—Electrical irritation of right vagus : coil 10.

P. 4 31 0.—(An open tube had been placed in trachea in the interval.) Chloroform on cap over the mouth of the tube. Effect kept up by artificial respiration till death ; heart stopped at 4h. 41m. 0s.

Q. 4 42 0.—Artery cut ; pressure fell to zero.

If the Fick tracings of Experiment No. 148 be compared with the photographic reproduction of tracings A and C of the Glasgow Committee, it will be seen that they are identical, and that the slow action of the heart, with great fall of pressure, which the Glasgow Committee attributed to some capricious action of chloroform upon the heart, was undoubtedly due to stimulation of the vagus from asphyxia. The tracings of the Glasgow Committee's experiments show nothing more than that chloroform lowers the blood-pressure, and that sometimes under chloroform the fall of pressure is sudden and irregular. When once it is proved, as it is by the experiments of the Hyderabad Commission, that there is no danger in either sudden or gradual falls of the blood-pressure, unless the heart is weakened by interference with its nutrition, the whole of the Glasgow Committee's contention, with regard to the danger of chloroform to the heart and the necessity for feeling the pulse during its administration, falls to the ground.

December 2nd.—No. 149.

Monkey of fair size. Temperature of the room 19 cent. Into chloroform box at 8h. 20m. 50s. Fallen down at 8h. 24m. 40s. Placed on the table at 8h. 25m. 40s. and kept quiet with chloroform. Left carotid ligatured and canula inserted into it. Right carotid looped. Trachea opened, and a glass tube connected with the India-rubber pump of the Junker apparatus and provided with an exit tube tied into it for artificial respiration. Connection with the manometers shortly before 8h. 54m.

One Ludwig and one Fick tracing.

H. M. S. *Observations.*

A. 8 55 30—Ligature of the right carotid.

B. 8 55 40—Struggling.

C. 8 57 30—Chloroform administration on a sponge held in front of the tracheal tube.

D. 9 0 40—Chloroform administration by letting the animal breathe through the Junker bottle without the India-rubber bellows attachment, and pushing chloroform in this way until respiration stopped.

E. 9 4 0—Artificial respiration in the ordinary way by compressing the chest.

F. 9 9 0—After recovery, artificial respiration continuously by means of the India-rubber bellows of the Junker apparatus attached to the tracheal tube, while measures were being taken to separate the head from the body.

G. 9 10 20—Slight hæmorrhage from a large artery.

H. 9 18 0—Slight hæmorrhage from the jugular vein.

I. 9 22 30—Pumping chloroform vapour through the Junker apparatus into the trachea until respiration ceased.

After the tracing ended, the head was severed, and the animal gave a few diaphragmatic gasps, but eventually died, the heart stopping finally at 9h. 35m.

December 2nd.—No. 150.

Full-sized pariah. Weight 30½ lbs. Into chloroform box at 11h. 10m. 55s. Fallen down at 11h. 21m. 50s. Placed on the table at 11h. 23m. 45s. and kept quiet with chloroform. Loop under both vagi and canula inserted into left carotid in the usual way. Connection made with the manometer at 11h. 41m. 15s. Three Ludwig and two Fick tracings (the 1st Fick reading is incorporated with the Fick of 149).

H. M. S. *Observations.*

A. 11 42 50—Smothering by holding the mouth and nose tightly closed. The effect of this is shown in Fick 2. The pulse fell from 116 before the smothering to 38 after.

B. 11 45 0—Gradual chloroform administration with plenty of air in the ordinary way.

H. M. S.

C. 11 48 30—Smothering again while the animal was well under chloroform.

D. 11 50 20—Smothering again when it was just about to come out of chloroform.

E. 11 53 45—Giving chloroform again for a short time and then at 11h. 55m. 15s. smothering the animal, during which proceeding respiratory movements completely ceased, and though a spontaneous attempt was made to breathe artificial respiration became necessary.

F. 12 4 5—Giving chloroform again with lots of air, and as soon as the animal's cornea was insensitive, smothering it.

G. 12 9 30—Giving chloroform with the cap crammed on to the muzzle so as to admit very little air, and pushing it until respiration ceased. Artificial respiration at 12h. 11m. 30s.

H. 12 17 35—Smothering again (*vide* Fick 7 and 8). These observations were accompanied by *very violent struggling*, and the smothering was consequently ineffective.

I. 12 23 15—Smothering again. The pressure fell 20mm. The effect of simple smothering in lowering the blood-pressure and slowing the pulse is well shown in the Ludwig tracing and in Fick 10; compare with Fick 2.

J. 12 37 30—Division of both vagi.

K. 12 38 20—Giving a little chloroform at times to keep the animal under.

L. 12 40 20—Smothering again. Fick 13 and 14 show that smothering after division of the vagi did not cause any slowing of the pulse as it did when the vagi were intact.

M. 12 48 0.—Inhalation of Amyl nitrite, of which, however, the quality was very doubtful.

N. 12 51 30.—Chloroform was then pushed until respiration ceased, and the animal died in spite of artificial respiration.

Before the last observation the animal's temperature had fallen below 95 F.

### December 2nd.—No. 151.

Temperature of the room 23½ Cent. Fair-sized pariah, very thin, and wounded. Weight 23 lbs. Into chloroform box at 3h. 14m. 3s. Fallen down at 3h. 20m. 5s. Placed on the table at 3h. 20m. 30s. Loop under both vagi. Temperature in the rectum at 3h. 26m. 101 F. Artery ligatured at 3h. 26m. 47s. Canula inserted at 3h. 27m. 55s. Connection with manometer at 3h. 44m. 30s. Two Ludwig and one Fick tracing.

*Observations.*

H. M. S.

A. 3 46 20—Ordinary chloroform administration at various times.

B. 3 50 30—Smothering by holding the mouth and nose. The blood-pressure fell 25mm. and the pulse dropped from 72 to 31 per minute.

C. 3 55 0—Cutting both vagi.

D. 3 58 25—Smothering again. The effect of smothering, now that the vagi are cut, is to cause an inappreciable fall of blood-pressure, and acceleration of the pulse to 105 per minute; compare Fick 2 and 10 of Experiment 150, and Fick 3 of 151, with Fick 5 and 6 of 151. The difference in the effects of smothering before and after division of the vagi is obviously due to the section of the nerves.

E. 4 1 40 -Artificial respiration.

F. 4 3 30 -Pushing chloroform until respiration ceased.

G. 4 8 10 -Inhalation of Amyl nitrite of somewhat better quality than in the last experiment.

H. 4 16 50—Chloroform again with struggling and eventual death in spite of artificial respiration. Temperature in the rectum at death 96 F.

### December 3rd.—No. 152.

Rabbit, weight 3¾ lbs. Temperature of the room 19 Cent. Chloroform at 7h. 52m. in a cubic foot box. Fallen down at 7h. 59m. 15s. Taken out of box at 7h. 59m. 45s. and kept quiet with chloroform. Canula inserted into left carotid. Connection with manometer a little before 8h. 35m. One Ludwig tracing and two Fick readings on the same tracing as 154. Chloroform was first given gradually and then pushed by pressing the cap closely upon the animal's nose and mouth. The effect of this latter proceeding is not shown in the tracing, owing to the artery becoming hopelessly drawn out of its sheath and twisted. Respiration ceased at about 8h. 52m. 30s. and the heart was still beating when the observation ceased at 9h.

### December 3rd.—No. 153.

Rabbit, weight 3¾ lbs. Temperature of the room 19·7 Cent. Into chloroform box at 9h. 45m. 40s. Fallen down at 9h. 52m. 40s. Taken out at once, but not being properly under, was put in again. The box was afterwards opened from time to time, but the animal was never fully insensitive. Convulsions began at 10h. 9m. 55s., and it was then taken out of box, and as it was not under, it was given more chloroform on a cap at once. It stopped breathing and artificial respiration was begun at 10h. 11m. 40s. Heart was still beating very rapidly, but efficiently when the thorax was opened at 10h. 14m. 45s. Auricle only beating at 10h. 52m. 30s. Auricle still beating regularly, though slowly, at 11h. 49m. Auricle still beating, but more feebly, at 12h. 7m. A portion of the auricle was still beating slowly at 1h. 2m., although all the organs had begun to shrivel up by drying.

### December 3rd.—No. 154.

Temperature of the room 20½ Cent. Monkey, fair-sized, but thin and wounded, weight 9¼ lbs. Into chloroform box at 10h. 44m. Fallen against the side at 10h. 51m. 15s., but still winking. Came out completely at 10h. 53m. 30s., and more chloroform was put into the box. Taken out of the box at 10h. 55m. 50s. and placed on the table and kept quiet with chloroform, while an operation to expose the spinal cord was performed. Temperature 102·8 F. in the rectum at 11h. 3m. Spinal cord exposed opposite the 5th cervical vertebra at 11h. 8m. Left carotid ligatured at 11h. 14m. 20s. Canula inserted at 11h. 15m. 43s. One Ludwig tracing and a part of one Fick (incorporated with 152).

#### *Observations.*

H. M. S.

A. 11 20 45—Ordinary chloroform administration.

B. 11 26 0—Chloroform by Junker's inhaler attached to the tracheal tube.

C. 11 27 15—Artificial respiration by the Junker bellows attached to the tracheal tube.

D. 11 30 30—Severance of the spinal cord at about the fifth cervical vertebra.

H. M. S.

E. 11 33 0—Chloroform again by Junker continued until the heart almost ceased beating.

December 3rd.—No. 155.

Fair-sized monkey, weight 9¾ lbs. Temperature of the room 23 Cent. Into chloroform box at 2h. 31m. 28s. Fallen down at 2h. 40m. Placed on the table and kept quiet with chloroform while the spinal cord was being exposed, during which proceeding there was a loss of about 1 oz. of blood. Temperature in the rectum 102 F. at 2h. 45m. Artery ligatured 2h. 51m. 45s. Canula inserted at 2h. 55m. 30s. Trachea opened and tube inserted at 2h. 57m. 50s. Connection made with manometer at 3h. 2m. A little chloroform was accidentally thrown into the trachea by the Junker apparatus and death resulted in spite of artificial respiration.

December 3rd.—No. 156.

Weight 27¾ lbs. A large pariah that has had two grains of phosphorus this morning. Into chloroform box at 3h. 26m. 30s. Fallen down at 3h. 39m. 35s. and placed on the board at 3h. 40m. 10s. Artery ligatured at 3h. 46m. 50s., and canula inserted at 3h. 48m. Loop under both vagi. Connection made with manometer at 3h. 51m. 7s. One Ludwig and two Fick tracings.

H. M. S. *Observations.*

A. 3 56 40—Administration of chloroform with a cap closely applied to the muzzle (Fick 2 shows the extreme inhibition of the heart's action probably owing to vagus stimulation from interference with the respiration).

B. 4 0 5—Gradual administration of chloroform with plenty of air.

C. 4 3 55—Simple smothering without chloroform by holding the mouth and nose (compare Fick reading 5 with Fick reading 2). These readings are identical (1) with each other ; (2) with Fick readings 4 and 9 of Experiment 117, which are tracings of simple vagus irritation ; (3) with Fick Reading 11 of Experiment 64, which is a tracing of vagus irritation and chloroform administration combined ; (4) with Fick Reading 13 of Experiment 178, which records the arrest of the heart, after stoppage of the respiration by chloroform poisoning ; and (5) with the Glasgow tracings A and C, more especially C.

H. M. S.

D. 4 7 0.—Division of both vagi.

E. 4 10 35.—Simple smothering again (compare the extremely rapid pulse in Fick 7, with the slow pulse of Fick 5 before the vagi were divided).

F. 4 14 50.—Chloroform again with the cap closely applied while the pulse was still extremely rapid (compare No. 151). The pressure fell rapidly, and after the chloroform was stopped the animal gave two or three convulsive gasps in rapid succession which had no effect on the pressure.

G. 4 16 20.—Artificial respiration failed to restore the animal and the heart stopped beating about six minutes after the last gasp. Temperature 101·4 F. Thorax opened at 4h. 23m. Heart quite still, but irritable.

December 4th.—No. 157.

A large pariah, weight 38 lbs., that had three grains of phosphorus yesterday—one in the morning and two in the evening. Temperature of the room 18½ Cent. Into chloroform box at 7h. 58m. 30s. Fallen down at 8h. 4m. 59s. Left carotid ligatured and canula inserted. Temperature 100·6 F. in the rectum. Both vagi looped. Connection with manometer at 8h. 26m. 40s. Two Ludwig and two Fick tracings.

*Observations.*

H. M. S.

A. 8 27 0.—Ordinary chloroform administration.

B. 8 28 15.—A remarkable fall of pressure and slowing of the pulse (*vide* Fick 2) that occurred after the chloroform had been stopped. The apparatus for holding the dog's muzzle was being removed at the time, but no exact cause could be assigned for the fall of pressure (which resembles that produced by asphyxia in other phosphorus dogs), except that the animal was holding its breath. After about a minute the pressure and pulse were restored to their previous condition without artificial respiration or other interference.

C. 8 35 0.—Snipping of the margin of the anus (Fick 3).

H. M. S.

D. 8 38 40.—Chloroform with the cap closely applied to the muzzle (*vide* Fick 4).

E. 8 42 45.—Chloroform again (the animal struggling and holding breath); pushed until respiration had stopped.

F. 8 44 0.—Artificial respiration.

G. 8 45 0.—Division of both vagi.

H. 8 48 20.—Chloroform very gently with lots of air until respiration had almost stopped.

I. 8 57 20.—Chloroform again pushed until the respiration ceased entirely.

J. 8 59 0.—Artificial respiration for broken periods. At one time the artificial respiration appeared to have the effect of preventing the natural rise of pressure (*vide* the Ludwig tracing just after Fick reading 9). The animal now entered into a completely anomalous condition in which he gasped slowly and irregularly and the pressure rose and fell in the most rapid manner without any apparent reason.

K. 9 10 40.—Irritation of the peripheral end of the right vagus, at first with the coils distant 15, and afterwards 10 centimetres, the latter proving effective.

L. 9 13 50.—Pushing chloroform until death resulted (the tracing was interrupted by clots in the tubes which were only cleared just before death).

December 4th.—No. 158.

Large pariah, weight 37 lbs., that had three grains of phosphorus yesterday like 157. Into chloroform box at 10h. 23m. 20s. Fallen down and at once placed on the table at 10h. 28m. 45s. Kept quiet with chloroform from time to time. Temperature 101 F. Breathing very feeble and slow at 10h. 31m. 30s. and artificial respiration performed now and again until 10h. 46m. Artery ligatured at 10h. 38m. and canula inserted. Connection with the manometers at 10h. 47m. Two Ludwig and one Fick tracing.

H. M. S. *Observations.*

A. 10 51 55.—Ordinary chloroform administration, struggling and holding the breath.

B. 11 4 0.—Pushing chloroform until respiration stopped.

H. M. S.

C. 11 8 10.—Injection of liquor atropinœ 10 minims into the peritoneum.

D. 11 12 5.—Irritation of the right vagus to test the effect of atropine.

E. 11 13 30.—Injection of 20 minims more liquor atropinœ into peritoneum.

F. 11 16 30.—Irritation of the right vagus again, with now a distinct rise of pressure. The vagi are paralysed by the atropine.

G. 11 21 0.—Pushing chloroform gently, but not without some struggling and holding the breath.

H. 11 29 0.—Smothering by holding nose and mouth (*vide* Fick 4, in which the pulse is alternately slow and quick according to the phase of respiration, but distinct vagus irritation is absent, and compare with Fick 2 and 5 of 156).

I. 11 31 0.—Pushing chloroform until respiration ceased; artificial respiration failed to restore the animal.

December 4th.—No. 159.

Temperature of the room 23·5 Cent. Dog, weight 35 lbs., that has had phosphorus (one grain yesterday, one to-day). Into chloroform box at 3h. 3m. 45s. Fallen down at 3h. 8m. 8s. and placed on the table at once and given chloroform from time to time to keep it quiet. Breathing stopped and artificial respiration performed at 3h. 14m. 35s. and continued for a few seconds. Temperature in rectum 101·9 F. Canula inserted into the artery at 3h. 17m. 10s. Connection with manometer at 3h. 19m. 8s. The animal was fully sensitive and chloroform was given almost immediately. Violent struggling with the breath held ensued and afterwards deep inspirations and howling, during which he must have inhaled chloroform very freely. Chloroform was stopped after one minute and respiration ceased after about three-quarters of a minute more. Artificial respiration was at once commenced, and after about 3½ minutes there were a few feeble gasping respiratory movements, which did not, however, raise the mean blood-pressure at all. Artificial respiration was continued for five minutes longer, but failed to restore the animal, the heart ceasing to beat about ten minutes after the chloroform was commenced. The liver was distinctly fatty, and the heart appeared to be soft and flabby. One Ludwig tracing only.

### December 4th.—No. 160.

Pariah dog, weight 24 lbs., has had two grains of phosphorus as in 159. Into chloroform box at 3h. 38m. 24s. Fallen down at 3h. 49m. 20s. Placed on the table at once. Canula inserted into the left carotid at 3h. 55m. 45s. Connection with manometer at 3h. 59m. 30s. One Ludwig tracing only. Chloroform was given at first very gently with plenty of air until the animal was fully under. The animal's head was then placed in a bladder which communicated with an apparatus (described in Appendix) for generating carbonic acid and the inhalation of this gas commenced. Blood-pressure fell slowly at first, but afterwards more rapidly. After 3¾ minutes the respiration stopped, and the bladder was at once taken off the dog's head and air freely admitted while artificial respiration was commenced. Gasping respiratory movements occurred after about a minute, but had little or no effect upon the blood-pressure, and ceased finally after another minute. Artificial respiration was resumed, but without avail. The liver was not apparently fatty.

### December 6th.—No. 161.

Temperature of the room 20 Cent. Large pariah, weight 26 lbs., that has had three grains of phosphorus in one-grain daily doses, but is not particularly sick, though out of a batch of seven, of which he is one, who were dosed with phosphorus in the same way, three died in the course of the day. Into chloroform box at 10h. 11m. 20s. More chloroform into the box at 10h. 17m. 40s. Fallen down at 10h. 22m. 40s. Placed on the table at 10h. 23m. 25s. Slight convulsions at 10h. 25m., after which the breath was held for about half a minute and artificial respiration was employed for a few seconds. More chloroform at 10h. 25m. 55s. as the animal was sensitive and groaning. Temperature in the rectum 100·4 F. Chloroform stopped again at 10h. 27m. 50s. More chloroform at 10h. 29m. 25s. Artery ligatured at 10h. 30m. Stop chloroform again at 10h. 30m. 10s. Canula inserted at 10h. 31m. 52s. More chloroform at 10h. 33m. 50s. until 10h. 34m. 30s. More chloroform again at 10h. 34m. 40s. Stop chloroform at 10h. 35m. 20s. Connection made with manometer at 10h. 36m. 30s. Three Ludwig and two Fick tracings.

| | H. M. S. | *Observations.* |
|---|---|---|
| A. | 10 39 25.— | Administration of alcohol by Junker's inhaler, the tube introduced into the nostril (the pressure in this animal fell in a marked way whenever he struggled). |

H. M. S.

B. 10 45 0.—Alcohol continued, but close fitting inhaler used in place of the tube in the nostril.

C. 10 51 30.—Chloroform administration with struggling.

D. 10 59 10.—Inhalation of carbonic acid, and chloroform at the same time (*vide* especially Fick reading 3).

E. 11 9 4.—Carbonic acid inhalation again (after giving fresh air), and for a short time chloroform as well (*vide* Fick 6).

F. 11 24 0.—Carbonic acid inhalation again with more careful exclusion of air. After the Carbonic acid was stopped he breathed spontaneously for a short time, but artificial respiration then became necessary at intervals.

G. 11 48 0.—Lastly, smothering by bandaging mouth and nose with an elastic band until he died.

December 6th.—No. 162.

Weight 9 lbs. Monkey that had half a grain of morphine at 12 o'clock hypodermically. Into chloroform box at 2h. 25m. 5s. Fallen down at 2h. 32m. 15s. and at once placed on the board. Artery ligatured and canula inserted as usual. Connection with manometer at 2h. 50m. 15s. Two Ludwig and one Fick tracing during repeated administrations of chloroform until respiration ceased, with an increasing interval on each occasion before commencing artificial respiration until at last it failed to restore life.

*Observations.*

H. M. S.

A. 3 52 55.—Gentle chloroform administration with plenty of air. At first there were irregularities in the respiration, sneezing for example, and the trace is slightly irregular. The respiration then became regular, and the fall of pressure was perfectly regular until cessation of the respiration at 2h. 58m. 0s. Artificial respiration was commenced at 2h. 58m. 30s. and stopped at 2h. 59m. 20s. The animal then went into a profound sleep until 3h. 10m. 35s., when more chloroform was given.

H. M. S.

B. 3 14 30.—More chloroform. The respiration stopped at 3h. 16m. 0s. Artificial respiration was commenced at 3h. 16m. 40s., and the animal was fully restored at 3h. 23m. 0s. The artificial respiration was stopped from 3h. 18m. 35s. to 3h. 19m. 0s., when Fick 6 was taken. There is barely any pulse tracing to be seen in Fick reading 6, but the heart was not weakened and the animal was revived completely by continued artificial respiration.

C. 3 27 5.—Chloroform more freely than before. Respiration stopped at 3h. 28m. 0s. and artificial respiration was commenced at 3h. 29m. 15s. Fick 9 was taken at 3h. 28m. 55s. and shows entire absence of pulse tracing, and yet, as in Fick 6, the heart was not weakened, and the animal was again rapidly restored by artificial respiration.

D. 3 38 40.—Repetition of B and C. This time the animal at first struggled and held its breath, and there was consequent irregularity of the circulation (*vide* the Ludwig tracing and Fick 11). The respiration stopped at 3h. 39m. 40s., and Fick 12 shows complete absence of pulse tracing again. Artificial respiration was commenced at 3h. 41m. 30s. and the animal was easily restored.

E. 3 48 10.—Chloroform pushed till death; the respiration stopped at 3h. 52m. 0s. and the heart stopped at 3h. 57m. 30s.

Experiment 162 shows that absence of the pulse tracing, which was observed every time the respiratory centre was paralysed, is no proof whatever that the heart has ceased to act, or, though it has ceased to act temporarily, that it has failed. It is remarkable that when the heart stopped (*vide* Fick 6, 9 and 12 and the Ludwig tracing on either side of these readings) there was no further fall of blood-pressure, showing that at the time of cessation the pressure was already as low as it could be. If direct weakening of the heart were the cause of the fall of blood-pressure in chloroform administration, we ought to find the heart-beats getting smaller and smaller up to the time when the pressure reaches its lowest, and gradually growing bigger and bigger again on the other side as the effect of the chloroform wears off. Instead of this, Experiment 162 shows that there were ample pulsations on both sides of the temporary pauses. If Fick 9, with the Ludwig tracing on either side, is carefully studied, it will be seen that up to the time the heart beats ceased, the pulsations recorded in the Ludwig were ample and strong. There was then a temporary arrest of pulsation for 6 seconds (*vide* Fick 9). Immediately after Fick 9 the pulsation returned for 5 seconds (*vide* the Ludwig between 3h. 28m. 0s. and 3h. 29m. 0s.) Afterwards there was a long pause of 20 seconds without any pulsations at all. This pause was so marked that Dr. Bomford thought a clot had formed in the tube, and at 3h. 29m. 10s. he wrote on the Ludwig tracing "? clot." Artificial respiration was just about to be commenced when the animal gave two spontaneous gasps, vigorous pulsations followed and the pressure was raised in five seconds to the height it was at before chloroform was administered in Observation C.

### December 7th.—No. 163.

Temperature of the room 23 Cent. Medium-sized pariah, weight 25¼ lbs. that has had three grains of phosphorus, one daily on the 3rd, 4th, and 5th instant. Is now very feeble and can hardly stand. Tied on to the dog-board at 10h. 44m. 15s. Given chloroform on the ordinary cap at 10h. 44m. 45s. Operation to tie carotid commenced at 10h. 45m. 30s. Chloroform stopped at 10h. 46m. 45s. Canula inserted into carotid at 10h. 50m. 15s. Temperature in rectum 94·6 F. Connection made with manometer at 10h. 55m. One Ludwig and one Fick tracing during very gradual administration of chloroform, the animal's head being inserted through an opening in the side of a large box (8 feet cubic contents) and carefully packed with wet cloths after one ounce of chloroform had been introduced into the box on blotting paper. Post-mortem temperature 94·8 F.

### December 7th.—No. 164.

Temperature of the room 23 Cent. Another phosphorus dog of the same batch, but does not appear to be particularly ill. Weight 29 lbs. Tied down on the board and given chloroform on a cap at 11h. 27m. 40s. Chloroform stopped at 11h. 29m. 6s. More chloroform at 11h. 30m. 45s. Stopped again at 11h. 31m. 6s. Both pupils dilated. Artery ligatured at 11h. 31m. 45s. Temperature in the vagina 102 F. Canula inserted at 11h. 34m. 6s. More chloroform at 11h. 36m. 10s. Connection made with manometer at 11h. 39m. 10s. Two Ludwig and one Fick tracing.

H. M. S. *Observations.*

A. 11 39 0.—Rapid chloroform administration while struggling and holding breath. Great irregularity is observed in the Ludwig tracing.

B. 11 41 0.—Gentle administration of chloroform in the ordinary way so as not to excite struggles. The contrast to Observation A is very marked.

C. 11 44 0.—Very gradual administration with the large box as in 163.

D. 11 52 40.—Snipping the skin of the thigh.

E. 11 53 50.—Excision of one of the nipples.

F. 11 55 10.—Evulsion of nails.

After these at 12h. 2m. 0s. the box was opened and the air freshened by means of a bellows and then closed again after another ounce of chloroform had been introduced. This was continued until the animal died. The respiration ceased at 12h. 12m. 0s., and the heart stopped at 12h. 18m. 30s. Post-mortem—Temperature 101 F. Liver very friable, but very congested and of a dark colour. Heart distended.

December 7th.—No. 165.

Monkey, weight 9 lbs., to whom one drachm of tincture of cantharides was given by the stomach yesterday evening and another this morning, and it does not appear to be ill at all. Placed on the board and given chloroform at 2h. 41m. 40s. Chloroform stopped at 2h. 45m. 30s. Chloroform again at 2h. 47m. 5s. for a short time. Again at 2h. 49m. Artery ligatured at 2h. 52m. 5s. and more chloroform at 2h. 52m. 30s. Stopped again at 2h. 53m. 10s. No urine in the bladder. Loop under the internal jugular vein. Canula inserted at 2h. 56m. A little chloroform again at 2h. 57m. 5s. One Ludwig tracing and one Fick reading on the same tracing as 167. Chloroform was given very freely on a cap closely applied while the animal was struggling. He then alternately held his breath and gave deep gasps while the pressure fell rapidly. Respiration stopped. Chloroform was discontinued and artificial respiration was performed. He afterwards breathed in a feeble snorting way for a short time and then stopped finally. Artificial respiration was again performed for several minutes, but without effect, the heart gradually ceasing. The bladder was empty and the kidneys congested. The mesentery was very much congested.

December 7th.—No. 166.

Unusually large pariah dog, weight 39 lbs. (unpoisoned, but old and toothless). Placed on the table struggling violently and given chloroform at 3h. 27m. 10s. Chloroform stopped at 3h. 28m. 50s. Struggling again and given more chloroform from 3h. 31m. 30s. to 3h. 33m. 10s. Artery ligatured at 3h. 33m. Noticed to have stopped breathing at 3h. 34m. 5s. and artificial respiration was performed until 3h 39m. A needle thrust into his heart at that time did not beat. Lungs pigmented and bloodless. Mitral valve almost cartilaginous on its free margin. This dog's death was the result of inattention on the part of the attendants, who were seeing how quickly they could tie the artery and were not watching the respiration.

December 7th.—No. 167.

Pariah, weight 38¾ lbs. (unpoisoned and healthy). Into chloroform box at 3h. 50m. 50s. Fallen down at 3h. 54m. 30s. and placed on the board at once. More chloroform at 3h. 55m. 45s. Stopped at 3h. 57m. More chloroform at 3h. 58m. Artery ligatured at 4h. 50s. Chloroform stopped at 4h. 1m. 15s. Artery ligatured and canula inserted. More chloroform at 4h. 3m. 35s. Stopped at 4h. 5m. 3s. Connection made with the manometer at 4h. 5m. 20s. One Ludwig and one Fick tracing (which includes the solitary reading of 165). After a little chloroform had been given 5mm. of nicotine were injected into the peritoneum. Shortly afterwards the pressure began to fluctuate violently and the dog to gape as if he were about to vomit. Fick reading 2 was then taken and immediately afterwards the pressure rose steadily and with slight oscillations to above 250 mm. and landed the marker on the top of the drum. There was no apparent cause for this rise and no spasm was noticed; but in the confusion it may have been overlooked. The pressure soon fell again to about 180mm. Chloroform was discontinued and respiration ceased for rather more than a minute. After another two minutes the pressure began to fall rapidly, and the administration of chloroform, which was continued for about half a minute, seemed to hasten this fall. The breathing soon after ceased and the animal died. Convulsive twitchings of the jaw, ears, and forepaws continued until after 4h. 30m. The movements continued for more than ten minutes after death, and were sufficiently forcible to jerk the handles of a pair of pressure forceps fixed on the end of the tongue off the table at each spasm.

December 9th.—No. 168.

Monkey, weight 9¼lbs., placed in the chloroform box at 10h. 19m. 44s. Fallen down at 10h. 28m. 42s. and at once placed on the table and kept quiet with chloroform. Artery ligatured at 10h. 36m. 47s. Canula inserted at 10h. 38m. 55s. Connection with the manometer at 10h. 42m. 45s.

One Ludwig tracing and part of one Fick (incorporated with 169 and 170).

*Observations.*

H. M. S.
A. 10 44 5.—Ordinary chloroform administration. No struggling; tracing regular.
B. 10 46 0.—Chloroform administration. Struggling and holding the breath; very irregular breathing continued with corresponding irregularity of the circulation until 10h. 53m. 30s.
C. 10 50 0.—Continued gentle administration of chloroform with plenty of air until death. The respiration stopped at 10h. 59m. 30s. and the heart at 11h. 3m. 0s. Post-mortem—Temperature 100·8 F.

### December 9th.—No. 169.

Dog, 37¾ lbs. Given chloroform at 11h. 12m. 30s. Fallen down at 11h. 15m. 55s. Placed on the table at 11h. 16m. 10s. More chloroform given 11h. 17m. 47s. Stopped at 11h. 18m. 25s. More chloroform at 11h. 19m. 5s. Artery ligatured at 11h. 19m. 55s. Stopped chloroform at 11h. 21m. 5s. Canula inserted at 11h. 21m. 21s. More chloroform at 11h. 22m. 35s. Stopped chloroform at 11h. 23m. 10s. Connection with manometer at 11h. 24m. 25s. One Ludwig and part of a Fick tracing during continued gentle administration of chloroform until death.

H. M. S.
A. 11 27 5.—Chloroform gently.
11 30 55.—Respiration stopped.
11 34 45.—Heart stopped.

### December 9th.—No. 170.

Weight 10¼ lbs. Monkey that has had a drachm of tincture of cantharides yesterday and the day before. Into chloroform box at 11h. 47m. 43s. Taken out and placed on the table at 12h. 5m. and kept quiet with chloroform from time to time. Artery ligatured at 12h. 9m. 20s. Canula inserted at 12h. 10m. 36s. Connection with the manometer at 12h. 13m. 10s. One Ludwig and a part of a Fick during gentle administration of chloroform until death ensued. Post-mortem—Temperature 104 F. Bladder quite empty. No visible congestion of the kidneys.

H. M. S.
A. 12 13 30.—Chloroform gently.
12 19 40.—Respiration stopped.
12 25 0.—Heart stopped.

### December 9th.—No. 171.

Rabbit. Weight 3 lbs. Placed on the rabbit-board at 2h. 43m. 50s. Given chloroform at 2h. 45m. and from time to time to keep it quiet. Artery ligatured at 2h. 54m. (very small). Canula inserted at 3h. Connection with the manometer at 3h. 5m. Part of one Ludwig tracing and of one Fick (incorporated with the tracings of 172).

*Observations.*

H. M. S.
A. 8 3 0.—Violent struggling.
B. 3 8 40.—Chloroform administration pushed until the respiration ceased.
C. 3 10 10.—Artificial respiration; the artery was then accidentally torn and the tracing concluded, as it did not seem as if the animal was coming round. The heart continued to beat until 3h. 17m.

### December 9th.—No. 172.

Another rabbit. Weight 3¼ lbs. Tied on the rabbit-board and given chloroform at 3h. 21m. 23s. Artery ligatured at 3h. 26m. 50s. Canula inserted at 3h. 29m. 10s. Connection with the manometer at 3h. 37m. One Ludwig tracing, and one Fick (incorporated with 171).

H. M. S. *Observations.*
A. 3 38 15.—Gentle chloroform administration on a sponge before the nose: struggling.
B. 3 43 15.—Chloroform again while struggling and holding the breath. (The pressure rose every time the animal held its breath.) Violent struggling.
C. 3 45 30.—Chloroform on a cap closely applied to the face with more struggling and holding of the breath.

H. M. S.

D. 3 48 10.—Chloroform on a cap very gently without exciting struggles, and pushed until respiration stopped, and the heart was very feeble. A kink in the tube stopped the tracing. The heart continued to beat until 4h. 9m.

It is excessively difficult to administer chloroform to rabbits without exciting struggling or making them hold their breath.

December 10th.—No. 173.

Large pariah dog. Weight 34¼ lbs. Chloroformed in the box at 10h. 27m. 38s. Fell down at 10h. 33m. 45s. Placed on the table at 10h. 34m. 2s. and kept quiet with chloroform. Artery ligatured at 10h. 38m. 25s. Canula inserted 10h. 39m. 45s. Connection with the manometer at 11h. 46m. 15s. One Ludwig and one Fick tracing.

H. M. S. *Observations.*

A. 10 47 50.—Ordinary chloroform administration until the cornea became insensitive twice.

B. 10 54 35.—Injection of one grain of cocaine into the peritoneum.

C. 10 57 50.—Chloroform again until cornea became insensitive.

D. 11 6 15.—After the animal had come round, chloroform again. very gently but continuously, until death. Another injection of one grain of cocaine. After the respiration had stopped two curious rises of pressure with improved action of the heart took place (*vide* Fick readings 17 and 19).

December 10th.—No. 174.

Medium-sized pariah. Weight 24¾ lbs. Temperature of the room 21½ Cent. Into chloroform box at 11h. 34m. 48s. Fallen down at 11h. 43m. 7s. Placed on the board at 11h. 43m. 20s. and kept quiet with chloroform. Artery ligatured at 11h. 48m. 10s. Canula inserted at 11h. 49m. 10s. Temperature in the rectum 102·8 F. Jugular vein ligatured at 11h. 57m. 10s. Canula inserted into the jugular at 11h. 58m. 45s. Connection with the manometer at 12h. 1m. 45s. One Ludwig and one Fick tracing.

H. M. S. *Observations.*

A. 12 2 20.—Chloroform administration while struggling and holding breath, but stopped almost immediately, after which the pressure fell considerably during a prolonged

H. M. S.

holding of the breath, but was restored to its former height by four deep slow inspirations, the cornea being still sensitive.

B. 12 5 30.—Chloroform again in the ordinary way three times, the third time until the cornea had been insensitive for a considerable time.

C. 12 15 40.—Chloroform again for a short time, and then at 12h. 17m. 0s. injection of five minims of nicotine into the jugular vein. After this there was an immediate fall of pressure with irregularity and slowing of the heart's action (*vide* Fick 10), then very rapid heart's action and rise of pressure, and convulsions followed by a very rapid fall. Death ensued four minutes after the nicotine was thrown into the heart.

December 10th.—No. 175.

Large goat. Weight 60 lbs. Thrown and given chloroform at 2h. 34m. 40s. Loop under both vagi. Artery ligatured at 2h. 41m. 40s. Canula inserted at 2h. 43m. 17s. Connection with the manometer at 2h. 50m. 15s. One Ludwig and one Fick tracing.

H. M. S. *Observations.*

A. 2 50 15.—Chloroform administration during struggling and holding the breath, which caused the usual fall in pressure. Chloroform was stopped after the pressure had risen again, and there was a well-marked after-fall.

B. 2 54 0.—Continued struggles and holding of the breath.

C. 3 0 0.—Chloroform again, very gently and without struggling, until the cornea became insensitive.

D. 3 6 50.—Chloroform gently and continuously until death. The respiration stopped at 3h. 11m. 0s. and the heart at 3h. 18m. 0s.

December 10th.—No. 176.

Smaller and rather thin goat, 46 lbs. Thrown and chloroformed at 3h. 28m. 30s. Artery ligatured at 3h. 43m. 45s. and canula inserted as usual. Connection with the manometer at 3h. 39m. 20s. One Ludwig and one Fick tracing during :—Continuous chloroform administration until death ensued with the cap closely pressed over the face. It shows the difficulty of killing an animal like the goat, which can persistently hold its breath for long periods. A very large quantity of chloroform was used.

December 11th.—No. 177.

Monkey. Weight 10 lbs. Into chloroform at 10h. 47m. 5s. Fallen down at 11h. 58m. 30s. Placed on the table at once and kept quiet with chloroform. Left carotid artery ligatured at 11h. 6m. 30s. Canula inserted at 11h. 9m. 20s., but the artery was accidentally cut across by the ligature and the canula could not be inserted again as the internal coats had collapsed. The left carotid was, therefore, tied above and below and abandoned. Right carotid artery ligatured at 11h. 18m. 7s. Canula inserted at 11h. 19m. 27s. Left vagus exposed and looped. Connection made with manometer at 11h. 26m. 5s. One Ludwig and one Fick tracing.

H. M. S. *Observations.*

A. 11 26 45.—Ordinary chloroform administration with struggles. (The trace is defective from kinks in the tube.)

B. 11 25 30.—Free chloroform administration while irritating the vagus. (The effect upon the vagus ceased before the electrodes were removed (*vide* Fick 5) and the chloroform was pushed until respiration ceased.)

C. 11 32 0.—Artificial respiration.

D. 11 36 0.—Injection of ether, one drachm, into the peritoneum, which had an unexpectedly depressing effect, probably from the dose being excessive or the locality chosen for the injection unsuitable. (?)

The experiment was interrupted by the canula breaking while being cleared and cutting the artery, and the animal was killed with chloroform.

December 11th.—No. 178.

Dog that had ½ a grain of morphine hypodermically at 12 o'clock and another ½ grain at 2 o'clock. Put into chloroform box at 2h. 1m. 5s. Fallen down at 2h. 6m. 30s. Placed on the table at 2h. 6m. 55s. and kept quiet with chloroform. Left carotid ligatured at 2h. 13m. 45s. Canula inserted at 2h. 14m. 52s. Connection with the manometer at about 2h. 18m. 30s. Three Ludwig and three Fick tracings. The object in this experiment was to ascertain if morphine had any effect in preventing the success of artificial respiration.

Chloroform was given four times gently until respiration ceased and longer intervals were allowed to elapse each time before artificial respiration was commenced. On the first occasion a remarkable slowing of the heart was noticed just before and at the time the respiration stopped (*vide* Fick readings 2 and 3). Artificial respiration was commenced after about half a minute and natural respiration was soon restored. On the next occasion the heart completely stopped at the same time as the respiration (*vide* Fick reading 8), but soon commenced to beat again. Artificial respiration was begun nearly two minutes after the cessation of natural respiration, but this was again speedily restored. On the third occasion the heart again stopped at the same time as the respiration (*vide* Fick 13) and did not resume its beating for more than half a minute. After more than two minutes the animal made two feeble gasps, but artificial respiration was not begun until four minutes after the original stoppage. The pressure rose almost at once and natural respiration soon returned. On the fourth occasion there was again marked slowing of the heart's action shortly after respiration had stopped (Fick 18). Artificial respiration was begun after two minutes and a half and was successful in restoring the natural respiration. After recovery the chloroform was again pushed rapidly and continued until death resulted without any attempt to restore the animal by artificial respiration. There was again some slowing and intermittence of the heart's action (*vide* Fick 26), but the heart continued to beat for eight minutes after the cessation of the respiration.

*Observations.*

H. M. S.

A. 2 19 45.—Chloroform. Respiration became very slow at 2h. 21m. 10s. At 2h. 22m. 0s. the pupils were both dilated and at 2h. 24m. 40s. the respiration stopped. This was immediately followed by a long cessation of the action of the heart; there was then one full beat and the very slow action which is recorded in Fick reading 3. It is to be observed that the cessation of the heart's action was not followed by any further fall of the blood-pressure, and that the pulsations on each side of the pause were ample and strong: in short, there was arrest, but there was no failure of the heart. Artificial respiration was resorted to at 2h. 25m. 25s., though there is no doubt the animal would have revived without it.

H. M. S.

B. 2 29 25.—Chloroform. At 2h. 33m. 0s. the respiration became very irregular, and ceased at 2h. 34m. 30s. At the same moment the heart stopped completely (*vide* Fick 8). It recommenced to beat very quickly, but there was a second and shorter arrest, recorded on the Ludwig tracing. Artificial respiration was employed at 2h. 36m. 30s., but it was unnecessary as the animal would have recovered without it.

C. 2 51 40.—Chloroform. The respiration was thought to have stopped at 2h. 53m. 40s., but did not actually stop till 2h. 54m. 45s. The heart stopped at the same moment for more than a minute. Breathing recommenced at 2h. 57m. 0s., but at 2h. 58m. 0s. the pressure fell again and artificial respiration was resorted to at 2h. 58m. 40. The blood-pressure rose rapidly and natural breathing recommenced at 3h. 1m. 0s.

D. 3 7 25.—Chloroform with plenty of air. The respiration stopped at 3h. 14m. 0s. Artificial respiration was commenced at 3h. 16m. 30s. and the animal soon breathed naturally, which he would probably have done without artificial respiration.

E. 3 28 0.—The animal was still stupified from the effect of the previous dose of chloroform when this observation was commenced. Chloroform was administered freely with a closely applied cap. Respiration stopped at 3h. 32m. 40s. and the heart ceased to beat at 3h. 40m. 25s.

Experiment 178 is very like Experiment 162 in which the heart's action was temporarily arrested every time the respiratory centre was paralysed. As in 162 there was no further fall of blood-pressure during the arrest, and the beats on each side of the stop were ample and strong. The most remarkable instance of this is seen at Fick 13. For more than 2 minutes there were only 17 very slight pulsations recorded, and for over a minute of this reading there was no pulsation at all. There was no fall of pressure during the arrest, and the Ludwig tracing shows how strong the beats on each side of it were. No better proof could be afforded than is to be found in these two experiments that direct weakening of the heart is not the cause of the fall of the blood-pressure which is inseparable from chloroform narcosis.

Experiments 64, 65, 162, 178 and 186 prove three important points :—

I.—A general fall of blood-pressure, whether sudden or gradual, is not in itself dangerous. (This is confirmed by the tracings of the Glasgow experiments).

II.—The fall of blood-pressure, which occurs in chloroformisation with regular breathing, is due solely to narcosis of the vaso motor system, and is, if not a safe-guard, absolutely harmless.

III.—The fall of the blood-pressure under chloroform is not due to weakening of the heart. The heart has nothing to do with producing it, unless the vagus is stimulated, or unles its nutrition fails either from imperfect oxygenation of the blood due to abnormal breathing, or from stoppage of the respiration from over-dosing.

December 13th.—No. 179.

Temperature of the room 20·5 Cent. Monkey small-sized. Weight 7 lbs. Chloroform given in box at 11h. 17m. Fallen down at 11h. 30m. 10s. and placed on the board. Artery ligatured at 11h. 40m. Trachea opened and tube inserted into it. Canula inserted into artery at 11h. 46m. Two Ludwig and one Fick tracings during—Continued very gradual administration of chloroform by dropping it into a funnel lined with blotting paper, which was connected by a tube with an opening in a large bottle. A second opening in the bottle was joined by a tube to the trachea, while a third opening was connected with a Marey's tambour registering on the slow Ludwig drum (*vide* Ludwig tracing).

The respiration stopped at 12h. 36m. 5s. and the heart ceased at 12h. 42m.

December 13th.—No. 180.

Medium-sized monkey, 9 lbs. Into chloroform box at 2h. 46m. Fallen down at 2h. 58m. 3s. and placed at once on the table. Trachea opened and tube inserted. Artery ligatured and canula inserted. Connection made with the manometer 3h. 14m. 45s. The experiment was prevented by the soda solution exuding out of the tracheal tube and killing the animal by convulsions and œdema of the lungs. The pressure in the manometer before the experiment was exactly that of the blood when connection was made (*vide* Ludwig tracing). The canula was correctly placed in the artery and had not cut it. The tracheal tube was also quite perfectly fixed. There was a lot of fluid in both pleural cavities. Lungs œdematous and one base carnified. Both the cavities of the heart contained a light coloured alkaline fluid. Peritoneum also full of similar fluid. The fluid from the trachea was strongly alkaline and effervesced freely on adding hydrochloric acid, and when tested with perchloride of mercury it gave a brown precipitate resembling that given by the soda solution when similarly tested. The only possible explanation of this case seems to be that communication had been accidentally made between the artery and the vein by which means the animal was injected with the soda solution.

December 13th.—No. 181.

Monkey, weight 7½ lbs. Into chloroform box at 4h. 2m. 45s. Fell down at 4h. 11m. 40s. Artery ligatured at 4h. 20m. 45s. Trachea opened at 4h. 23m. and a tube tied into it. Canula inserted into the artery at 4h. 25m. 20s. Chloroform at 4h. 27m. 45s. Connection with manometer at 4h. 30m. One Ludwig and one Fick tracing during gradual administration of chloroform through the same bottle as in 179. Respiration stopped at 4h. 44m. 45s. and the heart ceased acting at 4h. 52m.

December 14th.—No. 182.

Monkey, weight 9 lbs. Into the chloroform box at 7h. 42m. 40s. Fallen down at 7h. 51m. 10s. and at once placed on the table. Kept quiet with chloroform from time to time. Artery ligatured at 7h. 59m. 3s. Trachea opened and canula inserted at 8h. 3m. 30s. Canula inserted into the artery at 8h. 10m. 3s. Connection with manometer at 8h. 14m. Two Ludwig and one Fick tracing.

*Observations.*

H. M. S.

A. 8 16 20—Administration of alcohol, without any effect whatever.

B. 8 23 30—Administration of ether very gradually through the bottle apparatus used in 179.

Respiration ceased at 8h. 50m. 30s. Heart stopped at 8h. 56m. 0s.

## December 14th.—No. 183.

Dog, weight 30 lbs., that has had 2 grains of phosphorus this morning. Into chloroform box at 2h. 3m. 40s. Fallen down at 2h. 9m. 15s. Placed on the table and kept quiet with chloroform. Artery ligatured at 2h. 14m. Canula inserted at 2h. 16m. 40s. Connection made with manometer at 2h. 20m. 10s. Two Ludwig and three Fick[*] tracings.

### *Observations.*

H. M. S.

A. 2 20 45.—Chloroform administration on a saturated cap closely applied, during which the animal held its breath and an unusually well-marked fall of pressure occurred. The Fick apparatus was unfortunately not ready at this time. The chloroform was stopped as soon as the animal began to gasp to prevent any danger of over-dosing.

B. 2 23 25.—Chloroform administration in the same way repeated twice in the hope of obtaining a similar tracing on the Fick drum, but the animal did not hold its breath rigidly as in the first instance, and there was only an occasional slight slowing of the pulse (Fick 1 and 2).

C. 2 27 35.—Artificial respiration.

D. 2 31 30.—Salivation and some accumulation of fluid in the trachea.

E. 2 35 15.—Injection of 30 minims of liquor atropinœ into the peritoneal cavity.

F. 2 40 0.—Irritation of first the left and then the right vagus to show that the atropine had taken effect.

G. 2 45 30.—Smothering by holding the nose and mouth.

H. 2 51 0.—Chloroform again freely, but the animal neither struggled nor held its breath, owing probably to the obstructed condition of the trachea or to the weak state of the animal after phosphorus ; recovery was very slow.

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[*] Speed of the Fick drum increased in this experiment, so that it made a complete revolution in 1m. 9s.

H. M. S.

I. 3 3 25.—After the animal had come quite out and had begun to struggle, chloroform was again given very freely. The animal held its breath, but no marked slowing of the heart or fall of pressure occurred. Chloroform was finally pushed until death resulted.

December 16th.—No. 184.

Dog, weight 29 lbs., that has had two grains of phosphorus (one yesterday and one the day before) and was rather feeble in consequence. Chloroform given at 7h. 35m. 45s. Fallen down and placed on the table at 7h. 42m. Kept quiet with chloroform. Artery ligatured at 7h. 51m. Trachea opened and tube inserted. Canula inserted into the carotid at 7h. 59m. Connection made with the manometer at 8h. 5m. 30s. Two Ludwig and three Fick tracings.

*Observations.*

H. M. S.

A. 8 11 25.—Blowing up the chest forcibly by means of a bellows attached to the tracheal tube ; repeated three times.

B. 8 17 10.—Exhausting the air in the chest with bellows.

C. 8 24 40.—Production of asphyxia by stopping up the trachea ; repeated.

D. 8 37 0.—Injection of 30 minims of liquor atropinœ into peritoneal cavity.

E. 8 41 40.—Blowing up the chest again repeatedly.

F. 8 59 0.—Stopping up the trachea again. After the atropine, a fall of pressure still occurs in inflation of the chest, but no slowing of the heart (*vide* Fick 8 and 9). The slowing of the heart during asphyxia is also abolished. (Compare No. 158).

G. 9 1 40.—The animal was finally killed with chloroform.

December 16th.—No. 185.

Dog, weight 36 lbs., unpoisoned and healthy. Into the chloroform box at 2h. 30m. 35s. Fallen down at 2h. 36m. Placed on the table at 2h. 36m. 15s. Foaming at the mouth and kept quiet with chloroform. Artery ligatured at

2h. 42m. 30s. Canula inserted at 2h. 43m. 25s. Connection with manometer at 2h. 49m. 20s. Two Ludwig and five Fick tracings.

*Observations.*

H. M. S.

A. 2 51 0.—Struggling and repeated holding of the breath when he was quite out of chloroform.

B. 2 59 45.—Holding an ordinary cap with ammonia on it before the nose.

C. 3 1 20.—Chloroform administration during struggling and holding of the breath.

D. 3 5 20.—Snipping the anus.

E. 3 7 0.—Pulling out the tongue forcibly, which had the effect of making him hold his breath, and produced a fall of the blood-pressure and stoppage of the heart (*vide* Fick 5).

F. 3 11 0.—Chloroform during violent struggling.

G. 3 22 0.—Pushing chloroform until respiration stopped.

H. 3 31 20.—Squint operation. No effect.

I. 3 34 0.—Chloroform pushed until death resulted.

*N.B.*—The Fick drum was revolving in 1m. 9s. in this and the previous experiment. In all experiments before 183 the Fick revolution occupied 3m. 9s.

### March 6th, 1890—No. 186.

Healthy dog, weight 20 lbs. Temperature of room 28 Cent. Chloroform given in box 1h. 58m. 30s. Dog fallen down at 2h. 4m. Dog on table at 2h. 5m. Artery ligatured at 2h. 10m. 30s. Canula inserted at 2h. 12m. 30s. Connection with manometer made 2h. 16m. 30s.

*Observations.*

H. M. S.

A. 2 17 50.—Electrical irritation of entire vagus, coil 5, for 30 seconds. This observation shows the harmless effect of sudden lowering of the blood-pressure.

H. M. S.

B. 2 18 40.—Normal chloroform administration with regular respiration. The fall of the blood-pressure was perfectly regular and gradual. The breathing stopped at 2h. 24m. 0s., the vagus was stimulated at 2h. 24m. 15s. with the effect of slowing the pulse ; and the animal was restored without any other treatment.

C. 2 35 45.—Irritation of entire right vagus, coil 5, and simultaneous chloroform inhalation. The breathing stopped at 2h. 37m. 12s., and was restored without artificial respiration at 2h. 39m. 0s. The vagus stimulation was stopped at 2h. 39m. 5s. and the animal quickly recovered. The effect of the vagus stimulation was to suddenly lower the blood-pressure almost to zero, and chloroform was pushed while the pressure was low. If lowering the pressure were a danger with chloroform, this administration ought to have been particularly dangerous ; but the fall of pressure, with stoppage of the respiration, was obviously a safeguard, as the animal recovered without artificial respiration or any other measures being taken to revive it.

D. 2 48 25.—Repetition of Observation A for over a minute.

E. 2 53 32.—Chloroform inhalation with regular breathing. The respiration ceased at 2h. 56m. 0s. The entire right vagus was stimulated from 2h. 56m. 10s. to 2h. 57m. 25s. The breathing recommenced at 2h. 57m. 20s. and the administration of chloroform was continued for thirty seconds afterwards. The animal recovered without artificial respiration or other treatment. In this observation the stimulation of the vagus : after respiration had entirely stopped from overdosing with chloroform and the blood-pressure was very low : had the effect of still further lowering the pressure, and of keeping it nearly at zero, with a slow pulse, for over a minute. If the fall of pressure were in itself a danger under chloroform, the further fall ought to have increased the danger; but it proved to be a safeguard and saved the animal's life.

H. M. S.
F. 3 6 0.—Ordinary chloroform inhalation; interrupted to change the drum.

G. 3 13 40.—Ordinary chloroform inhalation pushed till the animal died. The breathing stopped at 3h. 15m. 50s.; there was slight vaso motor recovery at 3h. 18m. 0s.; the pulse began to fail at 3h. 19m. 25s.; a needle was inserted into the heart at 3h. 20m. 45s., and continued to move strongly and rhythmically until 3h. 23m. 0s. In this observation the animal was left entirely alone after the respiration ceased, and it died.

In Experiment 186 it might be said that the animal's recovery in Observation E was due to the vagus being entire, and to consequent stimulation of the respiratory centre when the nerve was irritated. But in the first place the respiratory centre was paralysed by chloroform, and until the effect passed off it could not respond to any stimulus; and in the second place, if the recovery was due to stimulation of the respiratory centre in Observation E, it must have been due to paralysis of the respiratory centre in Observation C, which is impossible. Observation E clearly shows that the fall of the blood-pressure under chloroform is not due to weakening of the heart. As in No. 178, the beats of the heart on each side of the inhibition were ample, and became smaller as the pressure rose during recovery, which could not be the case if the organ were weakened and the fall of the blood-pressure were due to this cause.

# INDEX TO MANOMETER AND OTHER TRACINGS, ON THE RECORDING DRUMS.

Abdomen—Pressure on ... ... ... Nos. 33, 88, 89.
Do. Bandage applied to ... ... „ 96, 97.
Do. Sudden blow on ... ... ... „ 79, 103, 104.
Do. Incisions into ... ... ... „ 83, 86, 104, 111, 116.
Do. Introduction of ice into ... ... „ 104.
A. C. E. Mixture.—Administration of ordinary—
A.—With a cloth cap ... ... „ 52.
B.—With Junker's inhaler—
1.—At regulated intervals ... „ 45, 46.
2.—Squeezing rapidly ... ... „ 45, 46.
3.—Through nostril lead tube ... „ 43.
Do. do. pushing of, until respiration had ceased entirely ... ... ... „ 49, 52.
Do. do. pushing until death results ... „ 45, 46, 47, 52, 55, 57, 62.
Do. do. in an asphyxiated animal... ... „ 62.
Acetic acid—Application to nostril ... ... „ 75.
Acid—Hydrochloric in saline solution introduced into the veins ... ... ... „ 93.
Alcohol—Inhalation of ... ... ... „ 161, 182.
Alkaline solution—Rapid absorption and effects of ... „ 179.
Amylintrite—Inhalation of ... ... ... „ 150, 151.
Ammonia—Inhalation of strong ... ... „ 35, 41, 65, 75, 76, 79, 95, 98, 99, 100, 111, 148, 162, 185.
Do. Injection into veins ... ... „ 111, 113, 116.
Do. Injection of aromatic spirit of—into stomach „ 116.
Antimony—Administration of ... ... „ 111.
Anus—Ligature of mucous membrane of ... ... „ 25.
Do. Introduction of thermometer ... ... „ 83, 103, 104, 119.
Do. Snipping of ... ... ... „ 25, 29, 31, 41, 62, 48, 104, 105, 157, 185.
Apomorphine ... ... ... ... „ 83, 86, 104.
Artificial respiration in the ordinary way ... ... „ 25, 32, 34, 36, 40, 41, 43, 48, 49, 52, 53, 60, 64, 65, 67, 70, 74, 76, 80, 81, 83, 84, 86, 87, 88, 89, 96, 97, 103, 104, 105, 106, 107, 111, 116, 117, 119, 141, 143, 144, 148, 149, 150, 151, 159, 160, 162, 178.
Do. do. by bellows apparatus... ... „ 119, 149, 154.

**Asphyxia** ... ... ... ... Nos. 56, 57, 58, 59, 60, 61, 63, 66, 70, 87, 89, 96, 97, 104, 105, 111, 113, 115, 135, 136, 138, 140, 148, 150, 151, 156, 157, 158, 161, 183, 184, 185.

Atropine ... ... ... ... „ 74, 76, 77, 80, 81, 86, (120), (121), 158, 183, 184.

Breath—holding of ... ... ... „ 39, 41, 51, 61, 103, 104, 164, 172, 174, 175, 176, 183, (*vide* also 184), 185.

Cantharides—Administration of ... ... „ 105, 170.

Carbonic acid—Inhalation of... ... ... „ 160, 161.

Carotid arteries, compressing both ... ... „ 70, 71, 95, 149.

Cats—Experiments on ... ... ... „ 138, 139.

Chloral ... ... ... ... „ 135.

Chloroform—Administration of—ordinary—

A—With cap ... ... ... „ 25, 29, 31, 32, 33, 34, 35, 36, 37, 38, 40, 56, 62, 64, 65, 69, 71, 74, 75, 76, 78, 79, 80, 81, 82, 83, 84, 86, 87, 96, 101, 102, 103, 104, 105, 107, 111, 113, 115, 116, 117, 118, 119, 133, 135, 136, 137, 139, 143, 144, 150, 151, 154, 156, 157, 158, 159, 160, 161, 162, 164, 167, 168, 169, 170, 171, 172, 173, 174, 175, 177, 183, 185.

B.—With Junker—

1.—At regulated intervals . „ 41, 43.

2.—Squeezing rapidly . „ 43.

3.—With lead tube in nostril . „ 43.

Do. Gradual administration in 8-foot box . „ 163, 164.

Do. Pushing of, until respiration ceased . „ 25, 29, 40, 41, 49, 53, 65, 71, 76, 78, 79, 80, 81, 82, 84, 86, 87, 88, 89, 97, 104, 105, 107, 111, 113, 116, 117, 118, 133, 136, 141, 143, 144, 148, 149, 150, 151, 152, 156, 157, 158, 162, 171, 172, 177, 178, 183, 185.

Do. Pushing, until death results... ... „ 25, 29, 30, 31, 32, 34, 35, 36, 40, 44, 48, 49, 53, 58, 60, 61, 63, 64, 67, 71, 74, 75, 76, 79, 80, 81, 82, 84, 91, 97, 101, 102, 103, 104, 105, 107, 111, 113, 116, 117, 119, 133, 135, 136, 139, 141, 145, 148, 154, 155, 157, 163, 164, 165, 168, 169, 170, 173, 175, 176, 178, 179, 180, 183, 184, 185.

Do. Direct introduction of, vapour into trachea „ 63, 67, 71, 98, 119, 135, 148, 149, 154, 184.

Chloroform—Direct introduction of, vapour into trachea through the Junker bottle ... No. 149.
Do. Direct introduction of, vapour into trachea through a large glass bottle ... Nos. 179, 181.
Do. Throwing, into trachea ... ... „ 64, 71, 155.
Do. Pushing, in an asphyxiated animal ... „ 58, 60, 61, 68, 97, 185, 136, 147, 148, 156, 158, 159.
Do. Pushing, during irritation of vagus ... „ 64, 65, 71, 103, 117, 118, 119, 137, 144, 177.
Do. Injection of, into jugular vein ... „ 72, 73, 91, 92.
Do. Sudden inhalation of concentrated vapour after holding the breath. „ 61, 103, 119, 185, and many others; *vide* "breath-holding."
Do. Sudden inhalation of concentrated vapour after artificial compression of the chest. „ 89.
Do. Inhalation of, with carbonic acid. „ 161.
Cocaine—Administration of— ... ... „ 173.
Cold—Application of cold cloths to the body ... „ 111.
Convulsions ... ... ... ... „ 105, 136, 167, 175.
Convulsive twitchings of muscles after death ... „ 167.
Coughing, produced by stimulation of vagus ... „ 78, 83, 95.
Deaths, accidental ... ... ... „ 48, 50, 54, 79, 85, 109, 110, 112, 135, 142, 153, 166.

Ether—Ordinary administration—
A.—On a cloth cap or open mouth-piece „ 34, 37, 40, 51, 55, 53, 69, 70, 95, 106, 108, 115, 116, 138, 140.
B.—With Junker ... ... „ 44.
C.—With bladder inhaler ... ... „ 43, 44, 45.
Do. Pushing of, until respiration ceased ... „ 40, 106, 108, 138, 140.
Do. Pushing of, until death results ... ... „ 33, 39, 51, 66, 70, 77. 95, 106, 108, 138, 140, 146, 182.
Do. Pushing together with irritation of vagus ... „ 119.
Do. Failure of, to keep the animal "under" ... „ 43, 44, 53, 55, 70, 89, 95, 108, 140.
Do. Direct introduction of, vapour into the trachea. „ 66, 70, 71, 89, 119, 131.
Do. do. do. through a large glass bottle ... ... „ 182.
Do. In an asphyxiated animal ... ... „ 61, 66, 70, 108, 138, 140, 148.
Do. Throwing, into trachea ... ... „ 70.
Do. Injection, into jugular vein ... ... „ 71, 72, 77, 93.
Do. Injection, subcutaneous ... ... „ 77.
Do. Intermittent inhalation of, for its stimulant effect ... ... ... ... „ 116.
Do. Injection into peritoneal cavity ... ... „ 177.
Exhaustion of the chest by bellows ... ... „ 184.
Eye-ball—Operation on— ... ... ... „ 84, 185.

Gasping ineffective after respiration had practically ceased ... ... ... ... No. 31, 34, 35, 64, 70, 74, 75, 76, 80, 82, 86, 92, 103, 104, 108, 111, 138, 139, 143, 159, 160.
Goats—Experiments on— ... ... ... „ 136, 137, 141, 143, 175, 176.
Hæmorrhage ... ... ... ... „ 32, 41, 113, 116, 149.
Head—Severance of— ... ... ... „ 149.
Heat—Application of hot cloths to the body ... „ 111.
Heart—Temporary stoppage of, at the same time as respiration ... ... ... „ 49, 60, 178.
Do. Stoppage of, before respiratory movement had ceased ... ... ... ... „ 80, 92, 103.
Do. Electrical stimulation of— ... ... „ 104.
Do. Introduction of needle into ... ... „ 51, 52, 53, 56, 57, 58, 59, 60, 63, 74, 75, 76, 86, 93, 103, 116, 141, 148.
Horses—Experiment on— ... ... ... „ 133, 142, 144.
Inflation of the chest forcibly by bellows... ... „ 184.
Intestine—Ligature of ... ... ... „ 25.
Larynx—Irritation of, with chloroform ... ... „ 104.
Monkeys—Experiments on—... ... ... „ 37, 68, 75, 78, 80, 82, 83, 86, 95, 96, 97, 98, 100, 103, 110, 111, 115, 116, 118, (123), (124), (125), (126), (127), (128), 129, (130), 131, 134, 135, 140, 149, 154, 155, 162, 165, 168, 171, 177, 179, 180, 181, 182.
Morphine—Administration of, before anæsthetic ... „ 47, 116, 162, 178.
Nails—Evulsion of— ... ... ... „ 25, 29, 31, 32, 37, 39, 40, 41, 79, 80, 164.
Nicotine—Administration of— ... ... „ 167, 174.
Nipple—Excision of— ... ... ... „ 164.
Nostril—Irritation of, Electrical ... ... „ 41.
Do. do. by chloroform ... ... „ 43, 75, 79, 95.
Operations—Others not specially entered—
 Jugular vein exposed ... ... „ 40, 76, 111, 116.
 Exposing femoral vessels ... ... „ 32, 41, 44, 45, 93, 103, 113, 116, 135.
 Phymosis ... ... ... „ 43, 79.
 Castration ... ... ... „ 103.
 Tracheotomy ... ... ... „ 64, 70, 71, 89, 119, 135, 148.
Phosphorus—Feeding with, previous to experiments ... „ 31, 81, 84, 104, 105, 106, 107, 108, 109, 119, 146, 147, 148, 156, 157, 158, 159, 160, 161, 163, 164, 183, 184.
Physostigmine—Use of ... ... ... „ 39, 40.
Position of body—Change of—to *vertical*... ... „ 31, 32, 79, 81, 83, 104.
Do. do. to *inverse* ... ... „ 33, 40, 79, 81, 106.
Rabbits—Experiments on— ... ... ... „ 99, 101, 102, (114), (120), (121), (122), 152, 171, 172.

Saline—Injection of, solution into veins ... ... Nos. 93, 103, 113, 116.

Sciatic nerve—Irritation of— ... ... „ 40, 67, 69, 70, 71, 95.

Skin—Snipping off a piece of— ... ... „ 164.

Soda bicarbonate introduced into the vessels accidentally. „ 91, 107, 136, 179.

Spinal cord—Severance of—(*see* Head) ... ... „ 154.

Splanchnic—Section of— ... ... ... „ 80, 103, 111, 115.

Spermatic cord—Division and irritation of— ... „ 103.

Stays—Imitation of—by plaster of Paris bandage ... „ 96, 97.

Stomach—Opening of—through abdominal wall ... „ 83, 104.

Do. Distension with air ... ... „ 83, 104.

Do. do. hot water ... ... „ 83.

Do. do. iced water ... ... „ 83.

Do. Pulling at—to imitate retching ... „ 104.

Do. Injection of spirits of ammonia into— ... „ 116

Struggling ... ... ... ... „ 74, 78, 80, 81, 82, 83, 84, 86, 88, 91, 97, 103, 104, 111, 113, 118, 119, 135, 138, 140, 143, 149, 157, 161, 164, 168, 171, 172, 174, 175, 177, 183, 185.

Sutures ... ... ... .. „ 83.

Teeth—Extraction of— ... ... .. „ 31, 40, 41, 79, 83, 95, 103, 104.

Testicles—Injury to— ... ... ... „ 80, 81, 84, 104.

Tetanus ... ... ... .. „ 167.

Tongue—Allowed to drop back ... .. „ 86.

Do. Pulling forward forcibly ... .. „ 185.

Ulna nerve—Irritation of— ... ... ... „ 83.

Urethra—Passages of instruments into—... ... „ 79, 103.

Vagus, *Right*—Irritation of the *entire*—

Mechanical ... „ 69, 70, 71, 78.

Electrical ... „ 35, 36, 39, 69, 74, 75, 76, 78, 80, 81, 87, 95, 103, 107, 108, 117, 118, 119, 148, 158, 183, 184.

By sodæ bicarbonas... „ 81.

By sodæ carbonas ... „ 36.

Vagus, *Right*—Irritation of the *peripheral* ... „ 36, 39, 64, 65, 69, 70, 71, 78, 95, 107, 135, 157.

Do. do. *central* ... ... „ 36, 41, 64, 65, 95, 135.

Do. Ligature of ... ... „ 36, 39, 41, 64, 65, 69, 70, 71, 78, 84, 95, 107, 108, 135.

Do. Division of ... ... „ 36, 39, 41, 64, 65, 69, 70, 71, 78, 82, 84, 88, 89, 95, 107, 108, 113, 115, 135, 150, 151, 156, 157.

Vagus, *Left*—Irritation of the *entire*—
Mechanical ... Nos. 39, 103.
Electrical ... „ 35, 37, 38, 39, 40, 69, 80, 95, 103, 118, 137, 144, 177, 183.
By sodæ bicarbonas... „ 35, 81.
By sodæ carbonas ... „ 36, 39.
Vagus, *Left*—Irritation of the *peripheral* ... ... „ 36, 95.
Do. do. *central* ... ... „ 36, 95.
Do. Ligature of ... ... ... „ 36, 64, 65, 69, 84, 95.
Do. Division of ... ... ... „ 36, 39, 64, 65, 82, 84, 88, 89, 95, 113, 150, 151, 156, 157.
Do. Peripheral—Irritation of both vagi simultaneously ... ... ... „ 64, 65.
Vomiting ... ... ... ... „ 104.

# PART VI.

## Experiments conducted by Committee.

| 1<br>Date. | 2<br>No. | 3<br>Description of dog. | 4<br>Time at which inhalation commenced. | 5<br>Cornea became insensible after | 6<br>Respiration ceased after | 7<br>Pulse stopped after | 8<br>Heart ceased beating after | 9<br>Artificial respiration commenced at | 10<br>Artificial respiration continued for | 11<br>Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|
| 1889. | | | h. m. s. | h. m. s. | h. m. s. | h. m. s. | h. m. s. | h. m. s. | h. m. s. | |
| Oct. 23 | 1 | Full-grown well-nourished pariah. | 2 51 0 | 0 2 0 | 0 3 20 | 0 3 55 | 0 3 55 | 2 54 55 | 0 3 0 | The chloroform administered freely on a cloth cap with a sponge inside. The respiration was in mere gasps for some time before its final cessation. The dog continued to yelp for some time after the cornea became insensible. Artificial respiration for three minutes failed to restore the pulse or respiration. P.-M.—Lungs healthy. Pulmonary veins much distended. Right auricle and ventricle full of blood. Left side empty. A leather muzzle was used to control the dog, which probably caused some constriction of the neck. Temperature of room 25·5° per cent. |
| Do. | 2 | Do. do. do. ... | 3 9 25 | 0 2 0 | 0 3 10 | 0 3 30 | 0 3 45 | Not employed. | ... | Chloroform administered as in No. 1. Leather muzzle as above. P.-M.—Conditions the same as in No. 1. |
| Do. | 3 | Do. do. do. .. | 3 26 0 | 0 0 43 | 0 1 50 | 0 3 23 | 0 3 45 | Do. | ... | Chloroform as in No. 1. In place of the leather muzzle a simple loop of rope was fastened round the jaws and tied behind the ears until the dog became quiet, so that no pressure was exercised upon the neck. The dog continued to yelp and groan for 47 seconds after the cornea became insensible. P.-M.—Both sides of the heart full of blood. Lungs normal. |
| Do. | 4 | Do. do. do. ... | 32 44 40 | 0 1 3 | 0 1 28 | 0 1 50 | 0 2 10 | 3 49 10 | 0 2 0 | Chloroform freely as in No. 1. The dog breathed more deeply than his predecessors. P.-M.—Right side of the heart full of blood; left side moderately so. Lungs normal. |
| Do. | 5 | Do. do. do. ... | 4 9 40 | 0 1 40 | 0 3 35 | 0 4 0 | 0 7 0 | 4 17 0 | 0 1 15 | Chloroform in ½-drachm doses on the same cap, five doses in all. Inhaled freely. Yelped a little after cornea became insensible. P.-M.—Lungs healthy. Right ventricle distended with venous blood. Left ventricle half full of bright arterial blood. |
| Do. | 6 | Do. do. do. ... | 4 24 10 | 0 1 10 | 0 5 13 | 0 5 55 | 0 7 5 | Not employed. | ... | Chloroform in ½-drachm doses, as in No. 5, five doses in all. Continued to yelp loudly after cornea became insensible. P.-M.—Conditions as in No. 5. |
| Do. | 7 | Full-grown well-nourished pariah, 2 hours after a full meal. | 9 45 0 | 0 0 50 | 0 2 55 | 0 4 15 | 0 6 50 | Do. | ... | Chloroformed freely in the ordinary hospital way. No muzzle used. Continued yelping 1m. 20s. after cornea became insensibe. One heaving respiration occurred about 30s. after the respiration was considered to have ceased. Veins of the brain full. Structure normal. Right auricle and ventricle distended. Left side almost empty. |
| Do. | 8 | Do. do. do. ... | 10 8 0 | 0 0 40 | 0 1 10 | 0 3 30 | 0 5 7 | Do. | ... | Chloroformed fully. No muzzle used. Respiration was thought to have ceased at 1m. 10s., but subsequently returned and continued until 3m. 20s. as noted in the table. The pulse similarly appeared to have ceased at 1m. 50s., but returned and continued |

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| | | | | | | | | | | until 3m. 30s. after the commencement of inhalation. No yelping or groaning. Both ventricles moderately firmly contracted. Right auricle distended. |
| Do. | 9 | Do. do. ... ... | 10 17 35 | 0 1 53 | 0 3 30 | 0 4 10 | 0 5 40 | Do. | ... | Chloroformed fully. No muzzle used. Respiration stopped after 2m. 40s., after which there were only three gasps, and it finally ceased at 3m. 30s. as noted in the table. Pulse stopped after 3m. 20s., but returned and continued for 4m. 10s. as noted. Groaning continued for 2m. 35s. |
| Do. | 10 | Full-grown pariah (light meal only at 7-30). | 10 9 40 | 0 5 25 | 0 9 50 | 0 11 25 | 0 13 53 | Do. | ... | Dog enclosed in a box 42½ in. by 19 in. by 17¼ in. (8 cubic feet contents), which was opened occasionally in order to examine the dog and chloroform given fully on a piece of blotting paper placed under the lid. No struggling or excitement. Removed from the box after 6m. and chloroform administered on a cap as before. Respiration ceased after 6m. 15s., but feeble occasional movements continued without probably any entrance of air into the chest for 9m. 50s. Heart as in previous cases. Blood in left ventricle rather venous in character. |
| Do. | 11 | Full-grown pariah just caught ... | 10 48 50 | 0 7 0 | 0 9 42 | 0 16 30 | 0 18 0 | 11 7 15 | 0 2 0 | Dog enclosed in the same box and chloroform administered as in No. 10. No excitement, the dog lying down quietly after about 4m. Removed from the box after 8m. 30s. and chloroform administered on cap as before, but the sponge was not pushed up close to nose so that the vapour was not so concentrated as in the previous cases; and the last air which entered the lungs therefore contained a less proportion of chloroform. Both ventricles full but not distended. The blood in the right ventricle dark; that in the left ventricle arterial, but not bright red. |
| Do. | 12 | Full-grown pariah, rather thin ... | 11 17 50 | 0 4 10 | 0 5 53 | 0 12 35 | 0 16 0 | 11 33 40 | 0 2 0 | Dog in box as before, but with a small opening covered with a glass lid in order to observe movements. No excitement. Removed from box at 4m. 30s. and chloroform administered on a cap as in No. 11. Chest opened after 14 minutes, so that heart's movements could be seen. Only flickering contraction for last two minutes, after which the trachea opened and lungs inflated by means of bellows, but no restoration of heart's movements occurred, although it was stimulated by pressure. |
| Do. | 13 | Full-grown pariah brought in 3 hours before, no food since arrival. | 11 40 50 | 0 4 28 | 0 5 50 | 0 7 7 | 0 8 30 | 11 48 40 | 0 1 50 | In box as before. No excitement. Dog fell down in box at 11h. 44m. 8s. Removed after 4m. 30s. and chloroform administered as before. Chest opened at 11h. 48m. 40s. The heart only feebly flickering for half a minute. |
| Do. | 14 | Full-grown but rather small-sized pariah dog, rather thin, no food. | 2 45 50 | 0 4 55 | 0 10 47 | 0 12 27 | 0 12 54 | ... | ... | The dog in the same box but excited by putting crackers inside the box and hammering it outside. Chloroform as before freely on blotting paper inside the box. Dog fell down after 3m. 45s. Removed from the box after 5m. 45s. and chloroform administered on a cap as before. Respiration very slow after 8 minutes. A few flickering movements of chest wall after respiration had practically ceased. Heart as in No. 11. Blood rather venous on left side. |
| Do. | 15 | Large-sized full-grown pariah, not fed recently. | 3 2 25 | 0 4 15 | 0 8 10 | 0 9 32 | 0 10 19 | ... | ... | The dog in box and excited by crackers as in No. 14. Dog fell down after 1m. 50s., but continued to struggle. Dog removed from box after 4m. 50s. and chloroform continued as before. Breathing very shallow after 7m. hardly more than flickering movement of the chest wall. P.-M.—condition the same. |

| 1<br>Date. | 2<br>No. | 3<br>Description of dog. | 4<br>Time at which inhalation commenced. | 5<br>Cornea became insensible after | 6<br>Respiration ceased after | 7<br>Pulse stopped after | 8<br>Heart ceased beating after | 9<br>Artificial respiration commenced at | 10<br>Artificial respiration continued for | 11<br>Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|
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| Oct. 24 | 16 | Small-sized but fully-grown pariah just brought in. | 3 17 50 | 0 6 55 | 0 15 50 | 0 17 40 | 0 18 11 | ... | ... | Dog in the same box, only without agitation. Chloroformed as in previous cases. Removed from the box after 7m. 5s. and chloroform continued as before. Evulsion of the nails performed after 9m. 17s. and while respiration continued. No apparent effect on the pulse in femoral artery. Internal rectus muscle cut through after 13m. 40s. Pulse did not intermit during operation but was felt to be hard and cordy. This condition had not been previously observed. Chloroform temporarily interrupted during operation. Pulse became feeble and intermittent after 15m. 5s.<br>A very little, almost venous, blood in the left ventricle. Right side full. Liver very much congested, the large veins of the splanchnic area engorged, but venous radicles almost empty. |
| Oct. 25 | 17 | Full-grown well-nourished dog fed with beef tea about a quarter of an hour previously. | 9 30 15 | 0 4 15 | 0 6 5 | 0 7 12 | 0 7 50 | ... | 9 34 5 | In the same box, but this had been made more air-tight. Chloroform fully on blotting paper. No excitement, the dog lying down after 3m. 50s. and rolling about from side to side. Convulsed violently at 4m. Taken out of the box at 4m. 45s. and chloroformed with the ordinary cap. Respiratory movements and heart sounds both feeble at that time. Post-mortem—heart structure normal. |
| Do. | 18 | Full-grown pariah ... ... ... | 9 49 20 | 0 1 22 | 0 2 30 | 0 4 0 | 0 4 22 | ... | ... | Chloroformed on the table with cap in the ordinary way. Pulse only flickering after 3m. 29s. No excitement and very little struggling. Heart healthy; both sides moderately full. That on left side rather venous in colour. |
| Do. | 19 | Full-grown well-nourished pariah. | 9 57 27 | Not observed. | 0 4 38 | 0 5 38 | 0 7 10 | ... | ... | In the box with 2 oz. chloroform on a towel put into the box with the dog. Dog fell down after 2m. 40s. and struggled violently. Respiration shallow after 3m. 30s. Taken out of box when pulse stopped. Heart healthy and in the same condition as the last. Liver very congested. |
| Do. | 20 | Full-sized well-nourished dog ... | 10 10 55 | Do. | 0 2 52 | 0 4 35 | 0 4 45 | ... | ... | Two oz. of chloroform introduced on blotting paper into the box and allowed to completely evaporate before the dog was put in. Sat down after 1m. Fell down after 2m., taken out after 3m. 45s. as the pulse had stopped, but it returned and flickered for nearly another minute. Heart as before; liver very congested; veins of the splanchnic, including the venous radicles, engorged. |
| Do. | 21 | Small badly-nourished pariah dog. | 10 27 25 | Do. | ... | ... | ... | ... | ... | Box prepared as in the last, but with only one ounce of chloroform. Dog introduced when the blotting paper appeared dry as in No. 20. Fell down after 1m. 40s. Struggling after 2m. Breathing laboured after 2m. 30s.; shallow after 3m. 35s. Jerking of the thoracic wall merely at 5m., but afterwards again breathing more regularly and deeply and lying quietly at the bottom of the box. Box opened at 11h. 46m. freely aired, and another ounce of chloroform placed inside on blotting paper. Cornea at this time quite insensible. When respiration had ceased, the dog was removed from the box, but no pulse could be felt or heart sound heard. |

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| Do. | 22 | Full-sized fairly-nourished pariah. | 10 50 33 | 0 12 25 | 0 19 24 | 0 21 30 | 0 22 15 | ... | ... | Chloroformed with Junker's inhaler. No muzzle. The ball being compressed 20 times in a minute. Struggled a little at first, cornea becoming insensible very gradually. Breathing ceased at 12m. 38s., but returned at 14m. 30s., the pulse during this interval being very rapid. Respiration then very shallow, but continued until 19m. 24s. after the commencement of inhalation Four drachms of chloroform used. Heart, liver and venous radicles as in No. 20. |
| Do. | 23 | Half-breed Spaniel-pariah badly nourished. | 11 27 33 | 0 2 32 | 0 5 18 | Not noted exactly. | 0 8 5 | ... | ... | Chloroformed with Junker at the rate of 40 squeezes per minute. The pulse stopped about ½m. after the respiration; exact time not noted. Two drachms of chloroform used. Left ventricle empty. Liver and venous radicles as in No. 20. |
| Do. | 24 | Half-breed black and tan pariah well nourished. | 11 47 20 | 0 1 27 | 0 7 32 | 0 7 49 | 0 9 0 | ... | ... | Chloroformed with Junker squeezing rapidly so as to keep the second ball distended. Respiration stopped at 4m. 26s., but the chloroform being discontinued to listen to heart, the respiration returned and finally ceased after 7m. 32s. Left side of heart almost empty and otherwise as in No. 23. 2½ drachms of chloroform used. |
| Do. | 25 | Full-sized strong pariah ... ... | 2 25 10 | ... | ... | ... | ... | ... | ... | Manometer experiment. |
| Oct. 26 | 26 | Full-sized pariah (brown) fairly nourished. | 7 44 0 | ... | 8 32 1 | 8 34 0 | No cessation. | Employed but not timed. | | Box 8 feet cubic contents, prepared as in No. 21, with eight drachms of chloroform fully evaporated before the dog was put in. Transferred at 8h. 19m. 30s. into another box of 9½ cubic feet contents prepared in the same way, before the dog was put into it, with 9½ drachms of chloroform, so that the proportion of the vapour was equal in the two boxes. Removed from the box at 8h. 32m. Heart beating very fast, but no pulse could be felt. Breathing returned after a few minutes but stopped again, and was only fully restored after artificial respiration had been employed. Dog fell down at 7h. 53m. Box changed at 8h. 19m. 30s. |
| Do. | 27 | Same dog ... ... ... ... | 8 52 0 | ... | 9 29 0 | 9 31 30 | 9 31 50 | Not employed. | | Repetition of the last experiment with the same dog, which was still drunk from the previous experiment. Recovered. Respiration returning about 1 minute after the heart was supposed to have ceased beating. Respiration again stopped 9h. 35m. 30s. Breathing again 9h. 37m. 30s. During this time pulse had not stopped. Cornea sensible at 9h. 38m. 30s. Respiration 34 per minute, and the dog fully recovered eventually. Was lying down at the commencement. Box changed at 9h. 22m. |
| Do. | 28 | Same dog ... ... ... ... | 10 44 0 | ... | 11 35 0 | 11 35 0 | ... | ... | ... | Same boxes as in the last with 8 drachms and 9½ drachms of chloroform, respectively. Respiration gasping at 10m. 48s. and afterwards continuously very feeble, but regularly. On respiration ceasing at 11h. 35m., he was taken out of the box and the pulse could not be felt and the heart, which was beating very slowly, was thought to have stopped. He recovered, however, without artificial respiration and commenced to breathe spontaneously at 11h. 41m. Dog fell down in box at 10h. 47m. 30s. Box opened at 11h. 23m. 30s. |
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[*] Nos. 29 to 185 were manometer experiments.

# Experiments conducted by Sub-Committee.

| 1<br>Date. | 2<br>No. | 3<br>Description of dog. | 4<br>Time at which inhalation commenced. | 5<br>Cornea became insensible after | 6<br>Respiration ceased after | 7<br>Pulse stopped after | 8<br>Heart ceased beating after | 9<br>Artificial respiration commenced at | 10<br>Artificial respiration continued for | 11<br>Remarks. |
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| 1889. | | | h. m. s. | h. m. s. | h. m. s. | h. m. s. | h. m. s. | h. m. s. | h. m. s. | |
| I (a).—*Large doses of chloroform given till death occurred, the chloroform being administered on a cloth cap inhaler. Dogs taken without any preparation.* | | | | | | | | | | |
| Oct. 24 | 186 | Full-grown pariah, healthy ... | 2 18 30 | 0 1 50 | 0 3 10 | 0 3 50 | 0 4 50 | ... | ... | Severe struggling and had to be held down forcibly for the administration. |
| Do. | 187 | Full-grown healthy pariah ... | 2 28 30 | 0 0 36 | 0 1 15 | 0 3 16 | 0 4 3 | ... | ... | Two gasps were made after respiration had ceased, but no air entered the lungs. Struggled severely and had to be held down forcibly. |
| Do. | 188 | Do. do. ... | 2 37 0 | 0 0 57 | 0 1 12 | 0 1 32 | 0 3 35 | ... | ... | Severe struggling and had to be held down forcibly. |
| Do. | 189 | Do. do. ... | 2 48 10 | 0 0 40 | 0 2 0 | 0 2 23 | 0 4 37 | ... | ... | Pulse returned for a few seconds before the heart stopped beating. Struggled a great deal. |
| Do. | 190 | Do. do. ... | 2 56 10 | 0 1 4 | 0 1 44 | 0 2 4 | 0 4 13 | ... | ... | Struggled severely. Gasped six times *after* the respiration ceased. |
| Do. | 191 | Under-sized pariah... ... ... | 3 8 50 | 0 0 37 | 0 1 15 | 0 1 23 | 0 4 20 | ... | ... | Struggled severely. |
| Do. | 192 | Pariah puppy, 9 months old ... | 3 16 10 | 0 0 36 | 0 1 37 | 0 1 56 | 0 5 23 | ... | ... | Struggled severely. A few flickering beats were felt at the pulse when the dog gasped, which he did once before the heart ceased beating. |
| Do. | 193 | Full-grown but small pariah ... | 3 23 4 | 0 0 48 | 0 3 50 | 0 5 48 | 0 8 27 | ... | ... | Struggled severely. Gasped twice before the heart stopped. |
| Do. | 194 | Full-grown pariah ... ... ... | 3 35 5 | 0 0 52 | 0 1 33 | 0 2 48 | 0 4 33 | ... | ... | Struggled. Gasped thrice before the heart stopped. |
| Do. | 195 | Do. ... ... ... | 3 42 8 | 0 0 56 | 0 2 42 | 0 3 32 | 0 5 28 | ... | ... | Struggled. |
| Oct. 25 | 196 | Do. ... ... ... | 9 49 0 | 0 0 50 | 0 1 25 | 0 2 4 | 0 3 42 | ... | ... | Struggled. |
| Do. | 197 | Do. ... ... ... | 10 6 4 | 0 0 46 | 0 1 13 | 0 2 25 | 0 4 19 | ... | ... | Gasped twice after the respiration ceased. Pulse stopped after 1m. 50s., and with gasping returned for 30s. Stopped finally after 2m. 25s. |
| I (b).—*Dogs fed on Liebig's Extract of Meat quarter of an hour before inhalation.* | | | | | | | | | | |
| Do. | 198 | Large-sized full-grown healthy pariah. | 10 12 8 | 0 0 51 | 0 2 52 | 0 3 44 | 0 5 15 | ... | ... | Had Liebig's Extract of Meat 2 teaspoonsful in hot water quarter of an hour before inhalation. Gasped 4 times and pulse returned after 40s. Pulse stopped again after 4m. 45s. Chloroform given as in the previous experiment. |
| Do. | 199 | Healthy large-sized pariah ... | 10 27 12 | 0 1 8 | 0 2 10 | 0 2 54 | 0 4 32 | ... | ... | Struggled. Had Liebig's Extract of Meat as in the above case. |
| Do. | 200 | Small-sized healthy pariah ... | 10 38 2 | 0 1 4 | 0 1 22 | 0 3 41 | 0 4 14 | ... | ... | Struggled. Gasped before heart stopped beating. do. |
| Do. | 201 | Full-grown well-nourished pariah. | 2 5 0 | 0 0 58 | 0 2 22 | 0 2 30 | 0 4 14 | ... | ... | Struggled. Had Liebig's Extract of Meat as in the last case. |
| Do. | 202 | Do. do. ... | 2 18 0 | 0 0 54 | 0 1 56 | 0 2 3 | 0 4 2 | ... | ... | Struggled. Had Liebig's Extract of Meat as in the last case. |
| Do. | 203 | Do. do. ... | 2 21 0 | 0 1 1 | 0 1 48<br>Gasped from 1m. 55s. till 2m. 9s. | 0 2 18<br>Pulse returned at 3m. 10s. till 3m. 37s. | 0 4 17 | ... | ... | Struggled. Respiration ceased after 1m. 48s. After 1m. 55s. gasped till 2m. 9s. Pulse ceased after 2m. 18s. Returned after 3m. 10s. and went on till 3m. 37s. when it finally stopped. Had Liebig's Extract of Meat as in the last case. |
| Do. | 204 | Full-grown well-nourished small-sized pariah. | 0 2 29 | 0 1 20 | 0 4 17 | 0 5 2 | 0 7 34 | ... | ... | Had Liebig's Extract of Meat as in the last case. |
| Do. | 205 | Full-grown well-nourished extra strong pariah. | 2 41 0 | 0 0 50 | 0 1 20 | 0 1 22 | 0 1 22 | ... | ... | In this case the dog, which was a very savage one, escaped twice before being chloroformed and struggled very forcibly. He had to be brought into the room with a tight rope and chain round the neck and was muzzled with the leather muzzle. The respiration ceased after 1m. 20s.; the muzzle and rope were removed |

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| | | | | | | | | | | as quickly as possible, but immediately afterwards, two or three seconds at most, the heart and pulse stopped simultaneously. It was impossible to perform ordinary artificial respiration, as no air could be forced in or out of the lungs.<br>Post-mortem—Lungs engorged, right and left ventrical of heart engorged, and distended with dark venous blood. Post-mortem appearances indicate death from asphyxia pure and simple. Liver contained less blood than usual, and it did not flow in section. Had Liebig's Extract of Meat as in the last case. |

I (c).—*Dogs kept fasting for 24 hours.*

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| Oct. 26 | 206 | Full-grown healthy pariah well nourished. | 8 30 0 | 0 1 47 | 0 2 50 | 0 3 22 | 0 7 44 | ... | ... | Struggled severely. Gasped several times in the interval between the stoppage of pulse and heart, and the pulse returned for 1m. 35s. (The chloroform in this and the following cases was given on the cloth cap inhaler.) |
| Do. | 207 | Full-grown pariah puppy, badly nourished. | 8 43 0 | 0 1 9 | 0 2 13 | 0 3 23 | 0 6 35 | ... | ... | Struggled severely. Gasped 5 times before heart stopped. Pulse returned for 30s. before heart stopped acting. |
| Do. | 208 | Full-grown well-nourished pariah | 8 54 0 | 0 1 40 | 0 3 0 | 0 8 0 | 0 8 48 | ... | ... | Struggled severely. Gasped several times before heart stopped. |
| Do. | 209 | Badly nourished, full-grown, small-sized pariah. | 9 9 10 | 0 1 7 | 0 3 4 | 0 4 6 | 0 6 23 | ... | ... | Struggled severely. Dog gasped 6 times before heart ceased to beat. |
| Do. | 210 | Ill-nourished, full grown pariah. | 9 21 0 | 0 1 33 | 0 3 43 | 0 4 20 | 0 9 16 | ... | ... | Struggled severely. |

I (d).—*Dogs that have had rectified spirit before inhalation. Chloroform in large doses on cloth cap inhaler.*

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| Do. | 211 | Full-grown healthy pariah ... | 10 42 0 | 0 0 50 | 0 3 2 | 0 3 48 | 0 6 18 | ... | ... | Struggled. Had ½oz. of spiritus rectificatus immediately before the inhalation. |
| Do. | 212 | Large-sized unusually strong pariah. | 10 58 0 | 0 0 44 | 0 2 34 | 0 2 54 | 0 5 4 | ... | ... | A small dose of chloroform was given preparatory to the administration of the spirit, as there was difficulty in getting him to swallow it without the anæsthetic. |
| Do. | 213 | Full-grown badly-nourished pariah with a healing wound on left side of thorax. | 11 15 0 | 0 2 22 | 0 3 51 | 0 4 15 | 0 6 28 | ... | ... | Had ½oz. of spiritus rectificatus with water 4 minutes before the inhalation. Little or no struggling. |
| Do. | 214 | Full-grown badly-nourished weak pariah (emaciated). | 11 24 10 | 0 1 33 | 0 8 23 | 0 9 15 | 0 12 46 | ... | ... | Had ½oz. of spirits and water 10 minutes before the inhalation, which made it drunk a minute after, and inhaled the chloroform quietly with little or no struggling. No force was required to compel this animal to inhale, and there was no holding of the breath as in the cases where no spirit was administered or it was administered immediately before the inhalation. |
| Do. | 215 | Full-grown well-nourished pariah. | 11 50 0 | 0 1 2 | 0 3 20 | 0 4 6 | 0 6 20 | ... | ... | This dog struggled a great deal although it had had ½oz. of spirits 10 minutes before the inhalation as in the last case. |

I (e).—*Five dogs that have had two teaspoonsful of Liebig's Extract of Meat two hours before the administration of chloroform in large doses.*

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| Do. | 216 | Full-grown healthy pariah ... | 2 30 0 | 0 0 42 | 0 1 22 | 0 1 38 | 0 4 18 | ... | ... | Struggled a great deal. Inhaler was covered by Mackintosh in this case to exclude air. Had Extract of Meat. The chloroform in this and the following cases was given as in the previous experiments. |
| Do. | 217 | Full-grown healthy pariah unusually strong. | 2 49 30 | 0 0 53 | 0 1 40 | 0 2 8 | 0 4 46 | ... | ... | Struggled a great deal. Pulse returned for 8s. after it had stopped. Had Extract of Meat. Chloroformed in the same way. |
| Do. | 218 | Full-grown ill-nourished pariah ... | 3 3 0 | 0 1 12 | 0 2 25 | 0 2 41 | 0 5 9 | ... | ... | Struggled severely. Pulse returned for 17s. before the heart stopped. Had Extract of Meat. Chloroformed in the same way. |
| Do. | 219 | Full-grown healthy well-nourished pariah. | 3 17 0 | 0 1 48 | 0 3 0 | 0 3 38 | 0 6 17 | ... | ... | Struggled severely. Had Extract of Meat. Chloroformed in the same way. |
| Do. | 220 | Large-sized powerful pariah. | 3 27 0 | 0 1 23 | 0 2 37 | 0 4 5 | 0 6 58 | ... | ... | Struggled severely. Gasped and the pulse returned for 40s. before the heart stopped. Had Extract of Meat. Chloroformed in the same way. |

| 1<br>Date. | 2<br>No. | 3<br>Description of dog. | 4<br>Time at which inhalation commenced. | 5<br>Cornea became insensible after | 6<br>Respiration ceased after | 7<br>Pulse stopped after | 8<br>Heart ceased beating after | 9<br>Artificial respiration commenced at | 10<br>Artificial respiration continued for | 11<br>Remarks. |
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| 1889. | | | h. m. s. | h. m. s. | h. m. s. | h. m. s. | h. m s. | h. m. s. | h. m. s. | |
| **I (f).**—*Five dogs that have had food two hours previous to inhalation and chloroformed in the above manner.* | | | | | | | | | | |
| Oct. 26 | 221 | Small-sized but full grown and healthy pariah. | 3 39 0 | 0 1 10 | 0 2 6 | 0 3 12 | 0 6 54 | ... | ... | Struggled. Gasped after pulse stopped, and the pulse returned for 15s. before the heart stopped. Had food two hours before. |
| Do. | 222 | Full-grown healthy pariah ... | 3 49 10 | 0 1 10 | 0 2 22 | 0 3 17 | 0 6 38 | ... | ... | Struggled very hard. Pulse returned for 28s. after it had stopped. Had food two hours before. |
| Do. | 223 | Lean but full-grown pariah ... | 4 10 54 | 0 1 4 | 0 1 43 | 0 3 5 | 0 4 36 | ... | ... | Struggled. Gasped after breathing had ceased. Had food 2 hours before inhalation. |
| Do. | 224 | Old and large-sized pariah... ... | 4 20 40 | 0 0 58 | 0 2 0 | 0 2 33 | 0 5 43 | ... | ... | Struggled a great deal. Pulse returned for 30s. after it had stopped. Had food two hours before. |
| Do. | 225 | Well-nourished full-grown pariah unusually strong. | 4 30 6 | 0 0 49 | 0 1 2 | 0 2 15 | 0 3 0 | ... | ... | Struggled very hard and was choked in being brought up to the table. Had food two hours before. |
| **I (g).**—*Dogs kept fasting from the previous evening chloroformed with large doses as usual.* | | | | | | | | | | |
| Oct. 28 | 226 | Full-grown well-nourished pariah. | 10 18 40 | 0 1 20 | 0 2 5 | 0 3 20 | 0 8 20 | ... | ... | Dog struggled very much during inhalation. Chloroformed fasting. The chloroform was given as in the previous cases on a cloth cap inhaler. |
| Do. | 227 | Under-sized fairly-nourished pariah | 10 32 8 | 0 0 57 | 0 1 32 | 0 2 2 | 0 6 42 | ... | ... | Dog did not struggle much. Chloroformed starving. |
| Do. | 228 | Large-sized full-grown powerful pariah. | 10 51 10 | 0 1 12 | 0 2 23 | 0 4 26 | 0 6 28 | ... | ... | Struggled hard. Gasped after pulse stopped. Pulse returned simultaneously for 40s. Chloroformed starving. |
| Do. | 229 | Large-sized full-grown pariah ... | 11 1 48 | 0 0 45 | 0 3 14 | 0 4 4 | 0 6 42 | ... | ... | Struggled hard. Chloroformed starving. |
| Do. | 230 | Large-sized full-grown but ill-nourished pariah. | 11 12 30 | 0 1 35 | 0 1 48 | 0 2 18 | 0 5 12 | ... | ... | Struggled hard. Gasped and pulse returned for 1 minute and 5 seconds before heart ceased contracting. Chloroformed fasting. |
| Do. | 231 | Badly-nourished full-grown pariah | 11 28 6 | 0 1 23 | 0 6 18 | 0 7 11 | 0 9 3 | ... | ... | Struggled a little. Chloroformed fasting. |
| Do. | 232 | Large-sized full-grown pariah ... | 11 45 15 | 0 2 6 | 0 2 49 | 0 3 13 | 0 5 12 | ... | ... | Struggled very hard. Gasped after respiration ceased. Pulse returned for 23 seconds. Chloroformed fasting. |
| Do. | 233 | Large-sized powerful pariah ... | 11 55 0 | 0 1 3 | 0 1 28 | 0 1 53 | 0 3 13 | ... | ... | Struggled very hard. Chloroformed fasting. |
| Do. | 234 | Full-grown pariah well-nourished. | 3 3 30 | 0 1 14 | 0 1 45 | 0 2 20 | 0 6 28 | ... | ... | Do. do. |
| Do. | 235 | Full-grown healthy well-nourished pariah. | 3 19 0 | 0 1 1 | 0 1 55 | 0 3 17 | 0 4 37 | ... | ... | Do. do. |
| Do. | 236 | Weakly pariah puppy 8 months old | 3 28 0 | 0 1 45 | 0 2 48 | 0 3 23 | 0 6 12 | ... | ... | Struggled feebly do. |
| Do. | 237 | Do. do. ... | 3 38 0 | 0 0 39 | 0 1 48 | 0 3 12 | 0 4 13 | ... | ... | Do. do. |
| Do. | 238 | Lean full-sized ill-nourished pariah | 3 47 45 | 0 0 57 | 0 2 14 | 0 3 26 | 0 5 27 | ... | ... | Struggled hard. Gasped several (13) times, when the pulse returned for 23s. Chloroformed fasting. |
| Do. | 239 | Lean and full-grown pariah pup about 9 months old. | 3 56 20 | 0 1 11 | 0 2 24 | 0 3 43 | 0 6 0 | ... | ... | Struggled. Chloroformed fasting. |
| Do. | 240 | Full-grown badly-fed pariah ... | 4 5 0 | 0 0 46 | 0 1 58 | 0 3 8 | 0 6 23 | ... | ... | Do. do. |
| Do. | 241 | Full-grown well-nourished pariah. | 4 13 6 | 0 2 3 | 0 2 23 | 0 3 8 | 0 5 0 | ... | ... | Struggled very hard. Gasped after breathing had stopped. Chloroformed fasting. |
| Do. | 242 | Large-sized full-grown powerful pariah. | 4 23 0 | 0 1 6 | 0 1 48 | 0 2 43 | 0 6 58 | ... | ... | Had to be muzzled. Struggled very hard. Gasped after breathing stopped, and the pulse returned for 12s. Chloroformed fasting. |
| Do. | 243 | Full-grown healthy pariah ... | 4 34 50 | 0 1 3 | 0 2 28 | 0 3 12 | 0 8 5 | ... | ... | Struggled feebly. Chloroformed fasting. |
| Do. | 244 | Full-grown well-nourished pariah. | 10 30 0 | 0 0 46 | 0 1 12 | 0 3 52 | 0 5 25 | ... | ... | Dog struggled. Chloroformed fasting. |
| Do. | 245 | Ill-fed pariah full-grown. | 10 37 55 | 0 0 38 | 0 1 22 | 0 2 33 | 0 4 53 | ... | ... | Struggled hard. Gasped. Chloroformed fasting. |
| Do. | 246 | Thin full-sized pariah dog with healing sore on back. | 10 43 10 | 0 0 56 | 0 1 14 | 0 3 37 | 0 6 0 | ... | ... | Struggled. Chloroformed fasting. |

**I (b).**—*Dogs chloroformed as they were obtained from the bazaars and chloroformed with large doses on cloth inhaler.*

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Do. | 247 | Full-grown badly-nourished pariah | 10 50 50 | 0 0 55 | 0 2 3 | 0 5 48 | 0 6 36 | ... | ... | Struggled hard. Gasped before heart stopped. Dog chloroformed as he was brought in. |
| Do. | 248 | Do. do. do. ... | 11 0 0 | 0 0 48 | 0 1 36 | 0 2 45 | 0 4 58 | ... | ... | Struggled. |
| Do. | 249 | Badly-nourished (emaciated) full-grown pariah. | 11 7 3 | 0 0 44 | 0 1 28 | 0 3 0 | 0 5 24 | ... | ... | Struggled. |
| Do. | 250 | Well-nourished full-grown pariah (very vicious). | 11 17 30 | 0 0 50 | 0 1 47 | 0 6 33 | 0 7 48 | ... | ... | Struggled very hard and gave trouble when being brought to the table. Gasped after breathing had stopped four times. |
| Do. | 251 | Full-sized ill-fed pariah ... ... | 11 26 0 | 0 0 36 | 0 0 53 | 0 1 51 | 0 3 17 | ... | ... | Struggled hard. |
| Oct. 29 | 252 | Full-sized well-fed strong pariah... | 11 32 40 | 0 1 22 | 0 2 31 | 0 4 16 | 0 11 12 | ... | ... | Struggled. In this experiment the cessation of the heart's action was judged by means of a needle thrust into that organ, and not by auscultation as in the former cases. |
| Do. | 253 | Emaciated full-grown pariah ... | 11 47 50 | 0 1 13 | 0 2 7 | 0 4 28 | 0 6 53 | ... | ... | Struggled. Gasped several times. Needle used as in the last case. |
| Do. | 254 | Full-sized healthy pariah .. ... | 3 1 11 | 0 1 59 | 0 2 42 | 0 3 58 | 0 12 8 | ... | ... | Struggled hard. |
| Do. | 255 | Old pariah blind of one eye from an opacity of cornea. | 3 15 30 | 0 0 37 | 0 1 35 | 0 3 36 | 0 4 38 | ... | ... | Struggled. Gasped after breathing had ceased. |
| Do. | 256 | Full-grown well-nourished pariah.. | 3 24 30 | 0 0 58 | 0 2 3 | 0 3 58 | 0 8 33 | ... | ... | Struggled. Gasped after breathing had ceased. Heart's action ceased to be heard with the stethescope after 4m. 37s., but on thrusting a needle into the heart, it was found to be acting and ceased only after 8m. 33s. from the time of inhalation. |
| Do. | 257 | Well-nourished full-grown powerful pariah. | 3 40 45 | 0 0 56 | 0 2 4 | 0 4 52 | 0 6 30 | ... | ... | Struggled. |
| Do. | 258 | Well-nourished full-grown pariah.. | 3 56 10 | 0 0 48 | 0 1 16 | 0 3 17 | 0 6 40 | ... | ... | Ditto. |
| Do. | 259 | Full-grown well-nourished pariah. | 4 4 0 | 0 0 53 | 0 1 50 | 0 2 43 | 0 7 38 | ... | ... | Ditto. |
| Do. | 260 | Full-grown ill-conditioned pariah. | 4 16 25 | 0 1 36 | 0 2 37 | 0 3 35 | 0 6 33 | ... | ... | Struggled very hard. |
| Oct. 30 | 261 | Full-grown pariah fairly nourished | 10 1 0 | 0 1 10 | 0 2 1 | 0 2 45 | 0 5 30 | ... | ... | Struggled and resisted as usual. Needle, with flag, thrust into the heart after stopping of pulse. Movements of flag, at first violent, gradually became feeble, and when they were reduced to mere vibration, the heart was said to have ceased. |

**II (a).**—*Two ounces of chloroform in tin box.*

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Do. | 262 | Ill-nourished middle-sized pariah.. | 10 13 30 | Not noted. | 0 45 28 | 0 48 15 | 0 52 12 | ... | ... | The anæsthetic was administered by placing the head and neck of the dog in a box 8 cubic feet in capacity, in which two ounces of chloroform had been evaporated. Dog struggled. Dog breathing naturally with a good pulse at 11h. 10m. Lid of box removed and 2 oz. of chloroform placed in it on blotting paper. Box covered again at 11h. 10m. 45s. The first dose of chloroform in this experiment was placed in the box an hour previous to the inhalation, and it was proved afterwards that the lid had been removed more than once during this time. |
| Do. | 263 | Ill-nourished under-sized pariah... | 11 30 0 | Do. | 0 0 45 | 0 2 30 | 0 3 45 | ... | ... | Box cleaned and dog chloroformed in the same way. Dog struggled. The inhalation was commenced immediately after the chloroform was put into the box. |
| Do. | 264 | Large-sized full-grown pariah ... | 11 43 0 | Do. | 0 1 46 | 0 3 30 | 0 5 0 | ... | ... | Dog struggled hard. Chloroformed in the same manner. |
| Do. | 265 | Ill-fed small pariah... ... ... | 3 0 0 | Do. | 0 2 43 | 0 6 8 | 0 6 55 | ... | ... | Struggled as usual. Chloroformed as in the last case. Gasped once before death. |
| Do. | 266 | Small-sized full-grown and weakly pariah. | 3 16 50 | Do. | 0 1 37 | 0 2 51 | 0 4 39 | ... | ... | Struggled as usual. Chloroform given as in the last case. |

**II (b).**—*One ounce of chloroform instead of two ounces given in the tin box as in II (a).*

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Do. | 267 | Full-sized healthy well-nourished pariah. | 3 31 15 | Not noted. | 0 1 48 | 0 3 2 | 0 7 38 | ... | ... | Dog struggled. One ounce of chloroform administered instead of two, but in the same tin box as in the last five cases. |
| Do. | 268 | Large-sized powerful pariah ... | 3 42 45 | Do. | 0 3 14 | 0 6 12 | 0 8 5 | ... | ... | Struggled a great deal. Chloroform given as in experiment 267. |
| Do. | 269 | Full-grown ill-nourished pariah ... | 3 55 0 | Do. | 0 2 35 | 0 4 26 | 0 5 8 | ... | ... | Struggled as usual. Chloroform given as in the last case. |

| 1<br>Date. | 2<br>No. | 3<br>Description of dog. | 4<br>Time at which inhalation commenced. | 5<br>Cornea became insensible after | 6<br>Respiration ceased after | 7<br>Pulse stopped after | 8<br>Heart ceased beating after | 9<br>Artificial respiration commenced at | 10<br>Artificial respiration continued for | 11<br>Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|
| 1889. | | | h. m. s. | h. m. s. | h. m. s. | h. m. s. | h. m. s. | h. m. s. | h. m. s. | |
| Oct. 31 | 270 | Full-grown ill nourished pariah .. | 9 48 0 | Not noted. | 0 2 30 | 0 6 30 | 0 7 42 | ... | ... | Chloroform in tin box as in the last case. Did not struggle at all during inhalation. |
| Do. | 271 | Full-grown well-nourished pariah. | 10 0 0 | Do. | 0 3 0 | 0 6 40 | 0 10 15 | ... | ... | Struggled as usual. Breathed again 50s. after the respiration ceased and continued breathing for 50s. Chloroform given as in the last case. |

**II (c).**—*Half an ounce of chloroform used in the tin box.*

| 1<br>Date. | 2<br>No. | 3<br>Description of dog. | 4<br>Time at which inhalation commenced. | 5<br>Cornea became insensible after | 6<br>Respiration ceased after | 7<br>Pulse stopped after | 8<br>Heart ceased beating after | 9<br>Artificial respiration commenced at | 10<br>Artificial respiration continued for | 11<br>Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|
| Do. | 272 | Fairly-nourished full-grown pariah. | 10 14 10 | Not noted. | 0 7 35 | 0 13 55 | 0 16 10 | ... | ... | Chloroformed as above. Struggled as usual, yelped loudly. Breathing returned 20s. after it had stopped and continued for 5m. 25s. afterwards. |
| Do. | 273 | Full-grown fairly-well-nourished pariah. | 10 35 20 | Do. | 0 19 4 | 0 19 6 | 0 19 12 | ... | ... | Struggled very hard and yelped loudly. The breathing stopped very gradually in this case, becoming shallow by degrees until it ceased. Chloroformed as in the last case. |
| Do. | 274 | Large-sized powerful full-grown pariah. | 11 2 0 | Do. | ... | ... | ... | ... | ... | Struggled and yelped loudly. Breathing became stertorous after 19m. 15s. Convulsions set in at 11-45 o'clock. The dog gradually recovered and was loose at 12-15. |
| Do. | 275 | Full-grown healthy pariah ... | 3 38 0 | Do. | 0 11 15 | 0 12 2 | 0 13 12 | ... | ... | Struggled and yelped loudly. Breathing stopped very gradually as in experiment 273. The action of the heart became very intermittent towards the end. There were three distinct intervals of about 8s. each, when there were no contractions, each interval being followed by very quick cardiac action. Chloroform given as in the last case. |
| Do. | 276 | Powerful full-grown healthy pariah. | 4 6 0 | Do. | 0 17 5 | 0 17 12 | 0 18 2 | ... | ... | Had to be muzzled. Struggled a great deal. Breathing ceased very gradually. Chloroformed as in the last case. |

**I (1).**—*Dogs chloroformed with about 2 oz. of chloroform at a time on the cloth inhaler.*

| 1<br>Date. | 2<br>No. | 3<br>Description of dog. | 4<br>Time at which inhalation commenced. | 5<br>Cornea became insensible after | 6<br>Respiration ceased after | 7<br>Pulse stopped after | 8<br>Heart ceased beating after | 9<br>Artificial respiration commenced at | 10<br>Artificial respiration continued for | 11<br>Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|
| Do. | 277 | Full-grown healthy pariah ... | 11 48 0 | 0 0 55 | 0 1 17 | 0 2 5 | 0 4 3 | ... | ... | Struggled as usual. |
| Do. | 278 | Do. do. ... | 11 55 0 | 0 1 7 | 0 1 58 | 0 3 21 | 0 5 34 | ... | ... | Do. |
| Do. | 279 | Full-grown pariah pup ... ... | 3 43 0 | 0 0 45 | 0 1 23 | 0 1 52 | 0 4 28 | ... | ... | Held its breath a great deal and then made some very full inspirations. Gasped before heart stopped. |
| Do. | 280 | Full-grown healthy pariah ... | 3 55 55 | 0 1 18 | 0 2 11 | 0 4 28 | 0 6 18 | ... | ... | The dog struggled as usual. |
| Do. | 281 | Large-sized full-grown pariah ... | 4 13 45 | 0 1 10 | 0 3 5 | 0 3 12 | 0 8 42 | ... | ... | Struggled in the usual manner. |

**II (a).**—*2 oz. of chloroform given in tin box.*

| 1<br>Date. | 2<br>No. | 3<br>Description of dog. | 4<br>Time at which inhalation commenced. | 5<br>Cornea became insensible after | 6<br>Respiration ceased after | 7<br>Pulse stopped after | 8<br>Heart ceased beating after | 9<br>Artificial respiration commenced at | 10<br>Artificial respiration continued for | 11<br>Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|
| Nov. 1 | 282 | Under-sized ill-fed pariah... ... | 9 49 0 | Not noted. | 0 4 30 | 0 6 0 | 0 7 24 | ... | ... | Chloroformed in tin box. Struggled as usual. Temperature in rectum at 9h. 53m. during inhalation 101·6 F. Temperature same after heart stopped. |
| Do. | 283 | Full-grown well-nourished pariah. | 10 2 30 | Do. | 0 11 47 | 0 12 30 | 0 15 17 | ... | ... | Did not struggle at all. Temperature in rectum at 10h. 5m. 30s. during inhalation 100 F. Temperature when heart stopped 100·5 F. |
| Do. | 284 | Under-sized ill-nourished pariah... | 10 26 30 | Do. | 0 4 40 | 0 5 25 | 0 5 30 | ... | ... | Chloroformed as above. Struggled as usual. Temperature in rectum before inhalation 102·4 F., when heart stopped 103 F. |
| Do. | 285 | Full-sized well-nourished pariah... | 10 41 50 | Do. | 0 4 46 | 0 5 52 | 0 9 38 | ... | ... | Temperature before inhalation 99·8 F. Struggled. Temperature immediately after death 100·4 F. |
| Do. | 286 | Full-sized pariah with opacity of cornea in one eye. | 10 58 13 | Do. | 0 4 24 | 0 5 8 | 0 8 59 | ... | ... | Temperature (*a*) before inhalation 102 F., (*b*) after heart stopped 102·8 F. Struggled. |

**II (b).**—*1 oz. of chloroform given in tin box.*

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Nov. 1 | 287 | Full-sized well-nourished pariah... | 11 12 0 | Not noted. | 0 8 31 | 0 10 0 | 0 14 6 | ... | ... | Struggled very hard. Temperature before inhalation 102 F., after heart ceased 103 F. |
| Do | 288 | Full-sized healthy pariah ... ... | 11 32 0 | Do. | 0 6 17 | 0 7 12 | 0 10 4 | ... | ... | Temperature before inhalation 103·8 F.; after heart ceased 104 F. Struggled as usual. |
| Do. | 289 | Emaciated full-grown pariah, lame in one leg from an old fracture. | 3 7 0 | Do. | 0 4 12 | 0 4 53 | 0 10 13 | ... | ... | Dog struggled. Temperature before inhalation 101·6 F. After death 101·8 F. Gasped before the heart ceased. Pulse returned and lasted for 1m. 50s. |
| Do. | 290 | Full-grown healthy pariah ... | 3 25 30 | Do. | 0 4 3 | 0 6 47 | 0 8 42 | ... | ... | Temperature before inhalation 103 F. Dog struggled. After death 103·4 F. |
| Do. | 291 | Ill-nourished full-grown pariah ... | 3 40 30 | Do. | 0 7 26 | 0 8 33 | 0 11 53 | ... | ... | Temperature before inhalation 103 F. Dog struggled. Breathing stopped very gradually. Temperature after death 104 F. |

**II (c).**—*Half an ounce of chloroform used in these cases in tin box.*

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Do. | 292 | Full-grown ill-nourished pariah ... | 3 58 15 | Not noted. | 0 8 11 | 0 9 38 | 0 10 28 | ... | ... | Dog struggled. Temperature before inhalation 103 F. After death 103·2 F. |
| Nov. 2 | 293 | Under-sized ill-nourished pariah ... | 9 26 0 | Do. | 0 10 40 | 0 11 10 | 0 15 40 | ... | ... | Struggled as usual. Temperature in rectum before inhalation 99·8 F. Temperature remained the same when heart ceased acting. Gasped at 9h. 38m. |
| Do. | 294 | Under-sized fairly-well-nourished pariah. | 9 47 12 | Do. | 0 16 0 | 0 17 25 | 0 19 55 | ... | ... | Struggled as usual. Temperature in rectum before inhalation 100 F., and remained the same when heart ceased acting. |
| Do. | 295 | Under-sized fairly-nourished pariah | 10 29 0 | Do. | 0 10 30 | ... | ... | ... | ... | Chloroformed as above. Temperature in rectum before inhalation 102 F. Respiration, after stopping for 30s., returned again, and the dog gradually recovered. Temperature when removed 102·6 F. Removed from the box at 11 o'clock. |
| Do. | 296 | Full-grown lean pariah ... ... | 11 7 45 | Do. | 0 14 8 | 0 16 31 | 0 18 1 | ... | ... | Temperature in rectum before inhalation 100·6 F. After death 101 F. Struggled a great deal. |

**III (a).**—*In these cases one-fourth of a grain of morophix hydrochloras was injected over the epigastrium of the dog 15 minutes before the inhalation. Chloroform in large doses given on the cloth inhaler.*

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Nov. 2 | 297 | Full-grown small-sized pariah ... | 11 14 30 | 0 1 3 | 0 1 45 | 0 2 7 | 0 6 33 | ... | ... | Morphine injected at 10h. 59m. Struggled as usual. |
| Do. | 298 | Full-grown well-nourished pariah.. | 11 50 0 | 0 1 38 | 0 3 12 | 0 3 37 | 0 6 22 | ... | ... | Morphine injected at 11h. 16m. Dog struggled. |
| Do. | 299 | Full-grown healthy pariah ... | 11 46 0 | 0 1 48 | 0 2 5 | 0 3 12 | 0 7 58 | ... | ... | Morphine injected at 11h. 32m. Struggled. Gasped after pulse stopped. |
| Do. | 300 | Full-sized well-nourished pariah puppy. | 2 36 0 | 0 0 35 | 0 1 5 | 1 1 10 | 0 2 5 | ... | ... | Morphine injected at 2h. 22m. Pulse ceased almost immediately after the respiration stopped. Struggled. |
| Do. | 301 | Full-sized well-nourished pariah ... | 2 45 0 | 0 0 43 | 0 1 33 | 0 1 52 | 0 5 3 | ... | ... | Morphine injected at 2h. 30s. Gasped after pulse stopped. |

**III (b).**—*Half grain morphine injected in these dogs before being chloroformed with large doses on the cloth cap inhaler.*

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Do. | 302 | Full-sized ill-nourished pariah ... | 3 3 10 | 0 1 1 | 0 2 12 | 0 3 4 | 0 5 17 | ... | ... | Morphine injected at 2h. 48m. Dog struggled. |
| Do. | 303 | Large-sized well-nourished pariah. | 3 16 2 | 0 0 44 | 0 2 8 | 0 3 36 | 0 4 53 | ... | ... | Morphine injected at 3 p.m. Struggled very hard and got loose. Caught and brought back and held down forcibly a second time. |
| Do. | 304 | Full-sized healthy pariah ... ... | 3 25 10 | 0 1 10 | 0 2 29 | 0 4 42 | 0 5 2 | ... | ... | Morphine injected at 3h. 11m. 20s. Gasped after pulse stopped. |
| Do. | 305 | Full-sized ill-nourished pariah with a cyst on tongue. | 3 38 20 | 0 1 3 | 0 1 36 | 0 2 28 | 0 5 47 | ... | ... | Morphine injected at 3h. 23m. |
| Nov. 4 | 306 | Full-sized fairly-nourished pariah. | 9 30 0 | 0 0 45 | 0 1 20 | 0 1 50 | 0 5 8 | ... | ... | Morphine at 9h. 15m Struggled as usual. Pulse stopped 30s. after respiration. |

**V (d).**—*Artificial respiration tried after respiration ceased on dogs that had had a subcutaneous injection of half a grain of morphine.*
*(The chloroform was given in large doses on the cloth cap inhaler.)*

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Do. | 30 | Under-sized fairly-nourished pariah. | 9 55 0 | 0 0 40 | 0 1 40 | 0 4 0 | Not noted. | 9 56 50 | 0 4 0 | Morphine injected at 9h. 38m. Pulse found to have stopped at 1m. after inhalation began. Artificial respiration commenced 10s. after respiration ceased; continued for 4m. Flag introduced into heart at the end of 4m. did not vibrate. Artificial respiration commenced 10s. after breathing stopped. Unsuccessful. |

| 1<br>Date. | 2<br>No. | 3<br>Description of dog. | 4<br>Time at which inhalation commenced. | 5<br>Cornea became insensible after | 6<br>Respiration ceased after | 7<br>Pulse stopped after | 8<br>Heart ceased beating after | 9<br>Artificial respiration commenced at | 10<br>Artificial respiration continued for | 11<br>Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | h. m. s. | h. m. s. | h. m. s. | h. m. s. | h. m. s. | h. m. s. | h. m. s. | |
| 1889. | | | | | | | | | | |
| Nov. 4 | 308 | Ill-nourished mangy pariah puppy between 4 and 5 months old. | 10 7 0 | 0 0 45 | 0 1 40 | 0 4 50 | 0 5 25 | 10 9 0 | 0 4 0 | Morphine injected at 9h. 47m. Struggled slightly. Artificial respiration continued for 4m.; no effect. Pulse stopped during artificial respiration. Artificial respiration commenced after 20s. Unsuccessful. |
| Do. | 309 | Full-grown pariah puppy emaciated. | 10 28 50 | 0 0 46 | 0 1 4 | Not noted. | Not noted. | 10 30 15 | 0 6 0 | Morphine injected at 10h. 17m. Dog died. Artificial respiration commenced after 21s. Unsuccessful. |
| Do. | 310 | Full-grown powerful pariah ... | 10 38 0 | 0 0 48 | 0 1 52 | ... | ... | 10 40 0 | 0 8 0 | Morphine injected at 10h. 23m. Natural respiration re-established at 10h. 48s. Artificial respiration commenced 8s. after breathing stopped. Successful. |
| Do. | 311 | Emaciated full-sized pariah ... | 11 6 0 | 0 1 0 | 0 3 4 | Not noted. | Not noted. | 11 9 20 | 0 12 0 | Half grain morphine injected at 10h. 52m. Artificial respiration commenced 16s. after breathing stopped. Unsuccessful. |
| **III (c.)**—*Half a grain of morphine injected 15 minutes before and varying quantities of strychnine immediately before administration of chloroform. Artificial respiration tried. Chloroform given in large doses in cloth cap inhaler.* | | | | | | | | | | |
| Nov. 5 | 312 | Ill-nourished under-sized pariah ... | 9 48 0 | 0 1 0 | 0 2 30 | 0 5 0 | 0 7 10 | ... | ... | Morphine injected at 9h. 33m.; ·02 gr. strychnine injected immediately before inhalation. |
| Do. | 313 | Badly-nourished under-sized pariah | 10 0 0 | 0 1 4 | 0 2 0 | 0 2 30 | 0 4 15 | ... | ... | Morphine injected at 9h. 45m.; and ·02 gr. strychnine immediately before inhalation. |
| Do. | 314 | Fairly-nourished small-sized pariah | 10 22 0 | 0 0 45 | 0 1 30 | 0 1 53 | 0 8 3 | ... | ... | Morphine injected at 10h. 7m.; ·02 gr. strychnine injected at 10h. 22m. |
| Do. | 315 | Full-sized well-nourished pariah... | 10 45 0 | 0 0 40 | 0 2 10 | 0 3 3 | 0 9 13 | ... | ... | Morphine injected at 10h. 30m.; strychnine (·02 gr.) injected at 10h. 44m. |
| Do. | 316 | Full-grown well-nourished pariah. | 10 54 0 | 0 0 46 | 0 1 23 | 0 2 28 | 0 6 8 | ... | ... | Morphine injected at 10h. 35m.; strychnine (·02 gr.) injected at 10h. 53m. |
| Do. | 317 | Full-grown small-sized pariah ... | 11 0 30 | 0 0 53 | 0 2 8 | 0 2 53 | 0 6 41 | ... | ... | Morphine injected at 10h. 44m.; strychnine (·02 gr.) injected at 11h. |
| Do. | 318 | Full-grown ill-nourished pariah ... | 11 8 0 | 0 0 42 | 0 1 7 | 0 2 40 | 0 6 1 | ... | ... | Morphine injected at 10h. 52m.; strychnine (·03 gr.) at 11h. 7m. 30s. |
| Do. | 319 | Full-grown healthy pariah ... | 11 16 30 | 0 59 0 | 0 2 1 | 0 2 32 | 0 6 30 | ... | ... | Morphine injected at 11h. 2m.; strychnine (·03 gr.) injected at 11h. 16m. |
| Do. | 320 | Full-grown healthy pariah ... | 11 25.12 | 0 1 0 | 0 3 30 | 0 4 11 | 0 9 33 | ... | ... | Morphine injected at 11h. 8m.; strychnine (·03 gr.) injected at 11h. 25m. |
| Do. | 321 | Full-grown healthy pariah ... | 11 36 30 | 0 1 20 | 0 3 46 | 0 4 15 | 0 9 50 | ... | ... | Morphine injected at 11h. 1m.; strychnine (·03 gr.) injected 11h. 35m. 50s. |
| Do. | 322 | Full-grown powerful pariah ... | 11 47 0 | 0 0 54 | 0 13 4 | 0 13 46 | 0 14 6 | ... | .. | Morphine injected at 11h. 21m.; strychnine (·03 gr.) injected 11h. 46m. The respiration ceased for 40s. at 11h. 52m. and returned.. |
| Do. | 323 | Full-grown healthy pariah ... | 12 6 0 | 0 1 15 | 0 2 0 | 0 2 54 | 0 4 50 | ... | ... | Morphine injected at 11h. 29m.; strychnine (·03 gr.) injected at 12h. 5m. |
| Do. | 324 | Full-grown large-sized healthy pariah. | 12 13 30 | 0 1 2 | 0 2 28 | 0 3 0 | 0 5 26 | ... | ... | Morphine injected 11h. 56m.; strychnine (·03 gr.) injected at 12h. 13m. |
| Do. | 325 | Full-grown large-sized ill-nourished pariah. | 2 59 0 | 0 0 45 | 0 1 15 | 0 2 10 | 0 3 40 | ... | ... | Morphine injected at 2h. 44m.; strychnine (·03 gr.) injected at 2h. 58m. Began to gasp 15s. after respiration ceased. |
| Do. | 326 | Under-sized ill-nourished pariah... | 3 4 0 | 0 0 45 | 0 2 25 | 0 2 30 | 0 6 40 | ... | ... | Morphine injected at 2h. 47m.; strychnine (1-10th gr.) injected at 3h. 4m. 15s. Gasped 5 times 25s. after cessation of respiration. |

III (d).—*Half a grain of morphine injected 15 minutes before the experiment and varying quantities of atropine immediately before. The chloroform was given in large doses on cloth inhaler.*

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Do. | 327 | Full-grown healthy pariah ... | 3 12 30 | 0 1 10 | 0 1 55 | 0 2 23 | 0 5 55 | ... | ... | Morphine injected at 2h. 50m.; atropine (1-100th gr.) injected at 3h. 12m. |
| Do. | 328 | Do. do. do. ... | 3 22 0 | 0 1 12 | 0 2 8 | 0 2 15 | 0 7 35 | ... | ... | Morphine injected at 2h. 55m.; atropine (1-50th gr.) injected at 3h. 21m. 30s. |
| Do. | 329 | Small-sized healthy pariah ... | 3 33 30 | 0 0 58 | 0 3 7 | 0 3 14 | 0 5 35 | ... | ... | Morphine injected at 3h. 10m; atropine (3-100th gr.) injected at 3h. 32m. |
| Do. | 330 | Small healthy pariah ... ... | 3 41 10 | 0 0 50 | 0 4 48 | 0 5 6 | 0 8 2 | ... | ... | Morphine injected at 3h. 20m.; atropine (1-25th gr.) injected at 3h. 40m. |
| Do. | 331 | Full-sized well-nourished pariah ... | 3 52 0 | 0 1 22 | 0 2 18 | 0 2 54 | 0 6 35 | ... | ... | Morphine injected at 3h. 35m.; atropine (1-20th gr.) injected at 3h. 51m. |
| Do. | 332 | Do. do. do. ... | 4 0 45 | 0 1 12 | 0 5 53 | 0 6 55 | 0 7 52 | ... | ... | Morphine injected at 3h. 46m.; atropine (3-50th gr.) injected at 3h. 59m. |
| Do. | 333 | Do. do. do. ... | 4 18 0 | 0 0 37 | 0 2 36 | 0 3 24 | 0 5 15 | ... | ... | Morphine injected at 3h. 55m.; atropine (7-100th gr.) injected at 4h. 17m. |
| Do. | 334 | Full-grown healthy pariah ... | 4 24 30 | 0 0 50 | 0 2 3 | 0 2 41 | 0 5 8 | ... | ... | Morphine injected at 4h. 5m.; atropine (8-100th gr.) injected at 4h. 23m. |
| Nov. 6 | 335 | Under-sized ill-nourished pariah ... | 10 8 0 | 0 1 20 | 0 2 25 | 0 2 30 | 0 5 5 | ... | ... | Morphine injected at 9h. 45m.; atropine (9-100th gr.) injected at 10h. 7m. Found to be quite narcotised from effect of morphine. |
| Do. | 336 | Do. do do. ... | 10 16 0 | 0 1 8 | 0 2 0 | 0 2 28 | 0 4 50 | ... | ... | Morphine injected at 9h. 55m.; atropine (1-10th gr.) injected at 10h. 15m. |

III (e).—*Half a grain of morphine injected some minutes before the experiment and varying quantities of atropine and strychnine immediately before. The chloroform was administered in large doses in cloth cap inhaler.*

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Do. | 337 | Full-grown fairly well-nourished pariah. | 10 41 0 | 0 0 55 | 0 1 30 | 0 2 42 | 0 4 0 | ... | ... | Morphine injected at 10h. 25m.<br>Atropine (1-100th gr.) } Strychnine (1-100th gr.) } injected at 10h. 19m. 45s.<br>Chloroformed with the cloth cap inhaler as in previous cases. |
| Do. | 338 | Full-grown under-sized fairly-well-nourished pariah. | 10 47 30 | 0 0 58 | 0 2 2 | 0 5 42 | 0 7 56 | ... | ... | Morphine injected at 10h. 33m.<br>Atropine (1-50th gr.) } Strychnine (1-50th gr.) } of each injected at 10h. 47m. |
| Do. | 339 | Full-grown large-sized pariah ... | 11 0 0 | 0 1 33 | 0 2 10 | 0 2 52 | 0 4 13 | ... | ... | Morphine injected at 10h. 45m. 30s. Injected strychnine (3-100th gr.) and atropine (3-100th gr.) at 10h. 59m. |
| Do. | 340 | Full-grown healthy pariah ... | 11 9 10 | 0 0 53 | 0 2 35 | 0 3 2 | 0 4 20 | ... | .. | Morphine injected at 10h. 55m. Injected strychnine (1-25th gr.) and atropine (1-25th gr.) at 11h. 7m. |
| Do. | 341 | Full-grown well-nourished pariah. | 11 28 5 | 0 1 28 | 0 2 5 | 0 3 3 | 0 6 15 | ... | ... | Morphine injected at 11h. 10m. Atropine (1-20th gr.) and strychnine 1-20th gr. injected at 11h. 27m. |
| Do. | 342 | Full-grown emaciated pariah ... | 11 40 0 | 0 0 59 | 0 1 30 | 0 2 3 | 0 4 0 | ... | ... | Morphine injected at 11h. 21m. Atropine (6-100th gr.) and strychnine (6-100th gr.) injected at 11h. 39m. |
| Do. | 343 | Large-sized full-grown pariah ... | 12 14 0 | 0 2 4 | 0 6 11 | 0 8 30 | 0 14 3 | ... | ... | Morphine (½ gr.) injected at 11h. 50m. Strychnine (7-100th gr.) and atropine (7-100th gr.) injected at 12h. 5m. Chloroformed in box as it attempted to bite every one that approached it. Lid of box removed and chloroform given in the usual way at 12h. 16m. 15s. |
| Do. | 344 | Full-grown ill-nourished pariah ... | 3 36 40 | 0 0 53 | 0 1 22 | 0 1 55 | 0 5 53 | ... | ... | Morphine injected at 3h. 20m. Atropine 8-100th and strychnine 8-100th gr. injected at 3h. 36m. |
| Do. | 345 | Do. do. do. ... | 3 45 0 | 0 1 8 | 0 1 46 | 0 2 12 | 0 8 12 | ... | ... | Morphine injected at 3h. 25m. Atropine 9-100th gr. and strychnine 9-100th gr. injected at 3h. 44m. |
| Do. | 346 | Do. do. do. ... | 3 55 0 | 0 1 6 | 0 1 48 | 0 2 0 | 0 5 6 | .. | ... | Morphine injected at 3h. 37m. Atropine 1-10th gr. and strychnine 1-10th gr. injected at 3h. 54m. |
| Do. | 347 | Small-sized healthy pariah ... | 4 15 45 | 0 1 4 | 0 2 1 | 0 2 48 | 0 6 3 | ... | ... | Morphine injected at 4h. 5m. Atropine and strychnine ā. ā. 1-10th gr. injected at 4h. 11m. |
| Do. | 348 | Full-grown pariah puppy ... ... | 4 21 0 | 0 1 13 | 0 1 24 | 0 2 0 | 0 4 53 | ... | ... | Morphine injected at 4h. 6m. Atropine and strychnine ā. ā. 1-10th gr. injected at 4h. 19m. 30s. |

| 1<br>Date. | 2<br>No. | 3<br>Description of dog. | 4<br>Time at which inhalation commenced. | 5<br>Cornea became insensible after | 6<br>Respiration ceased after | 7<br>Pulse stopped after | 8<br>Heart ceased beating after | 9<br>Artificial respiration commenced at | 10<br>Artificial respiration continued for | 11<br>Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|
| 1889. | | | h. m. s. | h. m. s. | h. m. s. | h. m. s. | h. m. s. | h. m. s. | h. m. s. | |
| | | **IV (a).**—*Chloroform administered in a special bottle and inhaler, with valves attached.* (Vide *Appendix B.*) | | | | | | | | |
| Nov. 7 | 349 | Small-sized badly-fed pariah ... | 10 47 0 | 0 10 0 | ... | ... | ... | ... | ... | This was a trial experiment to see if the valves in the apparatus worked properly. The dog was allowed to revive, as several interruptions occurred. |
| Do. | 350 | Full-sized healthy pariah ... | 11 13 30 | 0 6 43 | ... | ... | ... | ... | ... | Half an ounce of chloroform given in an apparatus specially devised for the purpose. By this means air was allowed to mix freely with the chloroform vapour in a bottle connected with the inhaler. Breathing became very rapid after the lapse of 14 minutes (62 to the minute), after 23m. it became very shallow and remained so for half an hour. The cornea remained insensitive for fully an hour and-a-half; the dog gradually recovered, and was removed from the table at 10m. past 1 p.m. |
| Nov. 8 | 351 | Full-grown-small-sized pariah ... | 10 4 0 | 0 0 27 | 0 44 45 | 0 45 5 | 0 47 10 | ... | ... | Half an ounce of chloroform given as in the above case. After 20m. the tube, connected with the inhaler, for the exit of the expired air, was removed, as the valves were acting imperfectly and the aperture of the tube was closed with a cork, to which a new valve was attached. Breathing became very slow and shallow towards death. This observation was not considered trustworthy on account of the interruptions during the experiment. |
| Do. | 352 | Small-sized ill-fed pariah ... | 10 37 17 | Not noted. | 0 7 3 | 0 10 14 | 0 10 34 | ... | ... | This observation is reliable, as the valves in the tubes were acting perfectly and no interruption occurred. ½ oz. of chloroform was given as in the above case. |
| Do. | 353 | Full-grown ill-nourished puppy ... | 11 14 30 | 0 1 48 | 0 18 8 | 0 19 1 | 0 22 14 | ... | ... | Half an ounce of chloroform given as in the last case. |
| Do. | 354 | Small-sized ill-fed pariah ... | 2 48 0 | 0 1 50 | 0 5 0 | 0 7 0 | 0 9 30 | ... | ... | Do. do. do. |
| | | **IV (b).**—*Three drachms of chloroform given in the same bottle apparatus.* | | | | | | | | |
| Do. | 355 | Small-sized ill-nourished pariah ... | 3 1 15 | 0 1 15 | 1 16 10 | 1 17 18 | 1 20 25 | ... | ... | 3 drs. of chloroform given in this case. The respiration became slow and laboured at 3h. 15m. and remained so for 45 minutes; but as there were no indications of the dog dying at this time, an extra drachm of chloroform was poured into the bottle at 4h. 3m. |
| Do. | 356 | Do. do. do. ... | 4 23 10 | 0 1 45 | 0 10 35 | 0 11 6 | 0 14 40 | ... | ... | 3 drs. of chloroform given as in the last case. After the breathing had stopped after 10m. 35s., it recovered in a minute's time and continued for 1m. and 8s., when it stopped again. |
| | | **IV (c).**—*Two drachms of chloroform given in the same bottle apparatus.* | | | | | | | | |
| Do. | 357 | Do. do. do. ... | 4 45 50 | 0 1 3 | ... | ... | ... | ... | ... | 3 drs. of chloroform given in the same apparatus as in the last case. An extra drachm was poured into the bottle at 5m. 20s. Was seen to be recovering at 5m. 49s. and an extra drachm of chloroform was again placed in the bottle. At 6m. 10s. the dog was found to be again reviving and he was removed from the table. |
| Nov. 9 | 358 | Do. do. do. ... | 9 48 20 | 0 2 20 | 0 20 10 | 0 23 30 | 0 25 10 | ... | ... | 2 drs. chloroform given as above, and had to be repeated as the dog was recovering at 10h. 8m. |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Do. | 359 | Full-sized well-nourished pariah ... | 10 18 6 | 0 1 30 | 2 7 30 | 2 8 40 | 2 9 2 | ... | ... | 2 drs. chloroform given as above. Dog was recovering at 11h. 9m. 1 dr. of chloroform added at 11h. 10m. Dog was seen to be recovering again at 12. when another drachm of chloroform was added. Dog 27 pounds in weight. |

**I (f).**—*Five dogs fed with gruel at 12-30 o'clock and chloroformed in the usual way with large doses on a cloth inhaler.*

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Do. | 360 | Full-grown healthy pariah ... | 2 30 5 | 0 1 8 | 0 2 35 | 0 3 28 | 0 5 2 | ... | ... | Struggled as usual. Weight of dog 24 lbs. |
| Do. | 361 | Do. do. ... | 2 41 20 | 0 1 2 | 0 1 53 | 0 3 47 | 0 5 11 | ... | ... | Struggled more than usual. Dog weighed 26 lbs. |
| Do. | 362 | Do. do. .. | 2 52 18 | 0 1 33 | 0 2 55 | 0 3 29 | 0 5 16 | ... | ... | Struggled as usual. Dog's weight 32 lbs. |
| Do. | 363 | Do. do. ... | 3 6 0 | 0 2 3 | 0 3 20 | 0 4 5 | 0 5 42 | ... | ... | Do. do. 28 lbs. |
| Do. | 364 | Do. do. ... | 3 15 30 | 0 0 42 | 0 1 56 | 0 3 55 | 0 5 6 | ... | ... | Do. do. 25 lbs. |

**IV (c).**—*Two drachms of chloroform given in bottle apparatus.*

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Nov. 11 | 365 | Full-grown well-fed pariah ... | 9 20 0 | 0 1 10 | 0 2 40 | 2 46 30 | 2 53 0 | ... | ... | 2 drs. of chloroform given in the bottle apparatus; 1 dr. repeated at 10h. 27m., and again at 11 and again at 11h. 40m. |

**I (f).**—*Five dogs fed with gruel at 9 a.m., and chloroformed with large doses on a cloth inhaler in the usual manner.*

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Do. | 366 | Full-grown large-sized powerful pariah. | 10 36 30 | 0 0 45 | 0 1 10 | 0 1 48 | 0 5 24 | ... | ... | Struggled as usual. Weight of dog 32 lbs. |
| Do. | 367 | Full-grown large-sized pariah ... | 10 46 30 | 0 0 54 | 0 1 38 | 0 2 5 | 0 4 40 | ... | ... | Do. do. 40 lbs. |
| Do. | 368 | Full-grown healthy and large-sized pariah. | 10 49 0 | 0 1 5 | 0 1 14 | 0 2 48 | 0 6 2 | ... | ... | Do. do. 34 lbs. |
| Do. | 369 | Full-grown healthy large-sized pariah. | 11 4 55 | 0 0 52 | 0 2 12 | 0 2 35 | 0 5 43 | ... | ... | Struggled more than usual. Weight of dog 32 lbs. |
| Do. | 370 | Full-grown strong pariah ... ... | 11 14 30 | 0 1 17 | 0 2 23 | 0 11 33 | 0 13 18 | ... | ... | Struggled as usual. In this case the respiration. after ceasing for 4m. during which period the pulse could be distinctly felt. returned again and lasted for 3¼ minutes. Weight of dog 38 lbs. |

**V (a).**—*Artificial respiration practised in these cases, the chloroform being given in large doses on the cloth cap inhaler.*

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Do. | 371 | Small-sized full-grown pariah ... | 11 39 30 | 0 1 10 | 0 2 40 | Had not stopped. | ... | 11 42 50 | 0 5 0 | Artificial respiration commenced 40s. after respiration had ceased. The dog was thought to be breathing naturally after one minute and was left alone. when the breathing ceased again and could not be re-established. Weight of dog 19 lbs. |
| Do. | 372 | Do. do. do. ... | 11 53 48 | 0 0 55 | 0 2 4 | Do. | ... | 11 55 59 | 0 2 0 | Artificial respiration was commenced 10s. after respiration had ceased. The dog recovered. Weight of dog 23 lbs. |

**I (l).**—*Monkey chloroformed in glass box.* (Vide *Appendix B.*)

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Do. | 373 | Small monkey ... ... ... | 1 58 43 | ... | ... | ... | ... | ... | ... | 2 drs. of chloroform into a one-foot cube glass case containing monkey; not air-tight: fell down after 2m. 15s., taken out after 9m. 50s. and chloroform pushed on cloth cap inhaler. Cornea sensitive; great salivation: lay on his side and grabbed at imaginary objects until 2h. 12m. 30s., when he jumped off the table. |
| Do. | 374 | Same ... ... ... ... ... | 2 12 55 | ... | 0 3 40 | Shortly after respiration. | 0 5 58 | ... | ... | Chloroformed in glass box and then taken out and the anæsthetic pushed on cloth cap inhaler. Monkey 4¼ lbs. in weight. |
| Do. | 375 | Small monkey ... ... ... | 2 21 30 | 0 1 30 | 0 2 0 | 0 2 10 | 0 9 30 | ... | ... | Chloroformed in the same way as before. Weight of monkey 5 lbs. |
| Do. | 376 | Do. ... ... ... | 2 34 0 | ... | 0 9 30 | 0 10 5 | 0 16 15 | ... | ... | In glass box at 2h. 34m. Fell down at 2h. 35m. 30s. Taken out of box at 2h. 38m. 30s. Cornea sensitive, but became insensible 30s. after removal from box and administering more chloroform. Heart ceased 6m. 10s. after respiration stopped. Weight 4 lbs. |
| Do. | 377 | Do. ... ... ... | 3 0 0 | 0 5 0 | 0 5 30 | 0 6 10 | 0 14 45 | ... | ... | In glass box at 3h. and chloroformed. Fell down at 3h. 4m. Taken out of box at 3h. 4m. 30s. and more chloroform given. Heart ceased 9m. 15s. after respiration stopped. Weight 5 lbs. |

| 1 Date. | 2 No. | 3 Description of dog. | 4 Time at which inhalation commenced. | 5 Cornea became insensible after | 6 Respiration ceased after | 7 Pulse stopped after | 8 Heart ceased beating after | 9 Artificial respiration commenced at | 10 Artificial respiration continued for | 11 Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|
| 1889. | | | h. m. s. | h. m. s. | h. m. s. | h. m. s. | h. m. s. | h. m. s. | h. m. s. | |
| Nov. 11 | 378 | Small monkey | 3 13 0 | 0 7 36 | 0 14 20 | 0 15 55 | 0 17 23 | ... | ... | In glass box at 3h. 13m. and chloroformed. Fell down at 3h. 20m. Taken out of box at 3h. 20m. 30s. Weight 5 lbs. |
| Do. | 379 | Do. | 3 35 0 | 0 0 32 | 0 0 50 | 0 1 20 | 0 3 21 | ... | ... | This monkey was asphyxiated when brought on the table owing to the noose around his neck having been drawn too tightly, and he was, after much difficulty, recovered. He was immediately after chloroformed on the table and not placed in the box. Weight of monkey 6 lbs. |
| Do. | 380 | Do. | 3 43 13 | 0 3 4 | 0 7 32 | 0 8 12 | 0 9 3 | ... | ... | Put into the glass box at 3h. 42m. 13s. Fell down at 3h. 45s. Taken out of box at 3h. 45m. 30s. ·Chloroformed pushed as in the other cases. |
| Nov. 12 | 381 | Young monkey | 10 23 0 | 0 8 30 | 0 11 30 | 0 12 50 | 0 14 15 | ... | ... | Put into the glass box at 10h. 23m., fell down at 10h. 30m. Taken out of box 10h. 32m. Temperature after taking him out of box 101·4 F., temperature after death 100·4 F. Weight 5 lbs. |
| Do. | 382 | Do. | 10 54 0 | 0 16 12 | 0 17 3 | 0 18 7 | 0 23 5 | ... | ... | Put into the glass box 10h. 54m. Fell down at 11h. 7m. 30s. Taken out of box 11h. 9m. 4s., and cloth inhaler placed over head. Temperature 103·2 F. when taken out of the glass box; after death 102·3 F. Weight 4 lbs. |

**I (j).**—*Five dogs chloroformed with large doses in cloth inhaler after the administration of a large quantity of coffee.*

| 1 Date. | 2 No. | 3 Description of dog. | 4 Time at which inhalation commenced. | 5 Cornea became insensible after | 6 Respiration ceased after | 7 Pulse stopped after | 8 Heart ceased beating after | 9 Artificial respiration commenced at | 10 Artificial respiration continued for | 11 Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|
| Do. | 383 | Full-grown small pariah | 11 42 0 | 0 0 45 | 0 1 42 | 0 2 52 | 0 4 15 | ... | ... | Temperature before inhalation 102 F. After death 103·2 F. Drank 12 oz. of prepared coffee at 11 o'clock. Weight 16 lbs. |
| Do. | 384 | Do. do. | 11 53 0 | 0 0 39 | 0 1 37 | 0 2 8 | 0 5 45 | ... | ... | Temperature before inhalation 104 F. Had 12 oz. of prepared coffee at 11. Temperature after death 104 F. Weight 18 lbs. |
| Do. | 385 | Full-sized badly-nourished pariah | 2 30 30 | 0 1 10 | 0 1 45 | 0 5 5 | 0 7 15 | ... | ... | Temperature before inhalation 100 F. Temperature after death 100·5 F. Had about 12 oz. of coffee about 2 minutes before inhalation. Weight 24 lbs. |
| Do. | 386 | Under-sized fairly nourished pariah | 2 43 0 | 0 1 15 | 0 2 30 | 0 3 50 | 0 6 35 | ... | ... | Temperature before inhalation 102 F. Temperature after death 102·8 F. Had about 12 oz. of coffee half an hour before inhalation. Weight 16 lbs. |
| Do. | 387 | Under-sized badly-nourished pariah. | 2 55 30 | 0 1 30 | 0 2 10 | 0 3 45 | 0 4 0 | ... | ... | Temperature before inhalation 102·5 F. Temperature after death 102·5 F. Had 12 oz. of coffee ¾ hour before inhalation. Weight 19 lbs. |

**V (a).**—*Artificial respiration tried in three cases, the chloroform being administered in large doses on cloth inhaler.*

| 1 Date. | 2 No. | 3 Description of dog. | 4 Time at which inhalation commenced. | 5 Cornea became insensible after | 6 Respiration ceased after | 7 Pulse stopped after | 8 Heart ceased beating after | 9 Artificial respiration commenced at | 10 Artificial respiration continued for | 11 Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|
| Do. | 388 | Full-grown large-sized pariah | 3 7 0 | 0 0 43 | 0 3 55 | ... | ... | 3 11 5 | 0 3 0 | Struggled a great deal. Artificial respiration commenced 10s. after respiration had ceased. Dog revived after artificial respiration had been practised for 2m. Weight 25 lbs. |
| Do. | 389 | Old large-sized pariah | 3 20 0 | 0 0 52 | 0 4 30 | ... | ... | 3 24 50 | 0 3 0 | Artificial respiration commenced 20s. after respiration had ceased. The dog gasped twice, but could not be revived. Weight of dog 32 lbs. |
| Nov. 13 | 390 | Full-grown small and badly-nourished pariah. | 10 48 0 | 0 0·46 | 0 1 30 | ... | ... | 10 49 50 | 0 3 0 | Artificial respiration was commenced 20s. after breathing had ceased. Dog revived. |
| Do. | 391 | Full-grown small-sized badly-nourished pariah. | 10 57 0 | 0 2 10 | 0 6 40 | ... | ... | 11 4 10 | 0 1 30 | Artificial respiration was commenced 30s. after breathing had ceased. Dog revived. |
| Do. | 392 | Full-grown well-nourished pariah. | 11 9 0 | 0 0 52 | 0 5 12 | ... | ... | 11 14 47 | 0 6 0 | Artificial respiration was commenced 35s. after breathing had ceased and proved unsuccessful. |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Do. | 393 | Old small and emaciated pariah ... | 11 31 0 | 0 1 10 | 0 2 25 | ... | ... | 11 34 0 | 0 4 0 | Artificial respiration was commenced 35s. after breathing had ceased and proved unsuccessful. |
| Do. | 394 | Under-sized fairly-nourished pariah | 2 40 0 | 0 0 45 | 0 1 30 | ... | ... | 2 42 0 | 0 6 0 | Artificial respiration commenced 30s. after respiration stopped. Gasped twice after 2m. and ceased. Artificial respiration started again and continued for 4m. and proved unsuccessful. |
| Do. | 395 | Full-sized well-nourished pariah ... | 2 50 0 | 0 0 55 | 0 2 5 | ... | ... | 2 52 20 | 0 9 0 | Artificial respiration commenced 15s. after respiration ceased and continued for 9 minutes. Proved unsuccessful. |
| Do. | 396 | Do. ... | 3 3 0 | 0 0 30 | 0 1 30 | ... | ... | 3 4 35 | 0 3 0 | Artificial respiration was commenced 5s. after the respiration had ceased. Dog revived. |
| Do. | 397 | Full-sized healthy pariah ... | 3 12 45 | 0 1 12 | 0 2 35 | ... | ... | 3 15 50 | 0 4 0 | Artificial respiration was commenced 30s. after the respiration had ceased and proved unsuccessful. |
| Do. | 398 | Do. ... | 3 27 30 | 0 1 17 | 0 2 45 | ... | ... | 3 30 50 | 0 1 0 | Artificial respiration was commenced 35s. after the respiration had ceased. Dog revived. |
| Do. | 399 | Small but full-grown pariah ... | 3 44 30 | 0 2 0 | 0 1 30 | ... | ... | 3 46 30 | 0 6 0 | Artificial respiration was commenced 30s. after the respiration had ceased. Unsuccessful. |
| Nov. 14 | 400 | Full-grown small ill-nourished pariah. | 10 26 0 | 0 0 7 | 0 8 8 | Not noted. | ... | 10 35 8 | 0 6 0 | Chloroformed in a dealwood box 8 cubic feet capacity. Dog fell down at 10h. 32m. 32s.; taken out at 3h. 33m. and placed on the table and some chloroform given. Artificial respiration was commenced 1m. after breathing had ceased. A needle was put into the heart at 10h. 41m. and the heart was found to be contracting. Artificial respiration continued for 6m., but proved unsuccessful. |
| Do. | 401 | Large-sized ill-nourished pariah ... | 10 53 0 | Not noted. | 0 12 0 | Not noted. | ... | 11 6 0 | 0 6 0 | Chloroformed in the same manner at 10h. 53m. Dog fell down at 10h. 57m. 30s. Taken out at 10h. 58m. and placed on the table and more chloroform given. Artificial respiration was commenced 1m. after the respiration ceased. It was continued for 6m., but proved unsuccessful. |
| Do. | 402 | Large-sized well-nourished pariah. | 11 15 0 | ... | 0 16 10 | ... | ... | 11 31 35 | 0 2 0 | Chloroformed in the same manner at 11h. 15m. Dog fell down at 11h. 23m. 45s. Taken out at 11h. 26m. 30s., placed on table and more chloroform given. Artificial respiration was commenced 25s. after the breathing had ceased and proved successful. |
| Do. | 403 | Do. do. ... | 11 36 0 | ... | 0 9 2 | ... | ... | 11 45 32 | 0 8 0 | Chloroformed in the same way. Fell down at 11h. 39m. 36s. Taken out and chloroformed on table at 11h. 40m. Artificial respiration was commenced 30s. after the breathing had ceased and proved to be unsuccessful. |
| Do. | 404 | Full-sized ill-nourished pariah ... | 11 53 10 | 0 3 30 | 0 6 8 | ... | ... | 11 59 43 | 0 4 0 | Chloroformed in the same way. Dog fell down at 11h. 55m. 28s. Taken out at 11h. 56s. placed on the table and chloroformed again. Artificial respiration was commenced 25s. after the respiration had ceased and proved unsuccessful. |
| Do. | 405 | Full-sized well-nourished pariah and one that had revived in a former experiment. | 12 7 0 | ... | 0 6 30 | ... | ... | 12 13 37 | 0 5 0 | Chloroformed in the same way. Fell down at 12h. 11m. Taken out of box 12h. 11m. Artificial respiration commenced 7s. after respiration had ceased, and proved unsuccessful. |
| Do. | 406 | Full-sized well-nourished pariah ... | 2 40 0 | ... | 0 0 5 | ... | ... | 2 45 20 | 0 9 0 | Chloroformed in the same box. Fell down at 2h. 43m. Taken out at 2h. 44m. 0s. and chloroformed on the table. Artificial respiration was commenced 20s. after respiration had ceased. Dog revived. |
| Nov. 15 | 407 | Under-sized fairly-fed pariah ... | 9 31 38 | 0 0 43 | 0 1 16 | ... | ... | 9 33 6 | 0 5 0 | Struggled a great deal. Artificial respiration was commenced 12s. after the breathing had ceased and proved successful. Chloroformed in large doses with cloth inhaler. |
| Do. | 408 | Full-grown well-nourished pariah.. | 9 40 30 | 0 1 2 | 0 2 0 | ... | ... | 9 42 40 | 0 4 0 | Struggled a great deal. Artificial respiration was commenced 10s. after breathing had ceased and proved successful. Chloroformed in large doses with cloth inhaler. |
| Do. | 409 | Full-grown small and well-nourished pariah. | 9 53 0 | 0 0 54 | 0 1 25 | ... | ... | 9 54 40 | 0 3 0 | Struggled as usual. Artificial respiration was commenced 15s. after the breathing had ceased and proved successful. Chloroformed in large doses with cloth inhaler. |

| 1<br>Date. | 2<br>No. | 3<br>Description of dog. | 4<br>Time at which inhalation commenced. | 5<br>Cornea became insensible after | 6<br>Respiration ceased after | 7<br>Pulse stopped after | 8<br>Heart ceased beating after | 9<br>Artificial respiration commenced at | 10<br>Artificial respiration continued for | 11<br>Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|
| 1889. | | | h. m. s. | h. m. s. | h. m. s. | h. m. s. | h. m. s. | h. m. s. | h. m. s. | |
| Nov. 15 | 410 | Under-sized lean pariah ... ... | 10 13 0 | 0 1 0 | 0 2 4 | ... | ... | 10 15 19 | 0 4 0 | Struggled a great deal. Artificial respiration was commenced 15s. after the breathing had ceased and proved successful. Chloroformed in large doses with cloth inhaler. |
| Do. | 411 | Small full-grown well-nourished pariah. | 10 33 30 | 0 0 45 | 0 2 40 | ... | ... | 10 36 30 | 0 2 0 | Struggled as usual. Artificial respiration was commenced 20s. after the breathing had ceased and proved successful. Chloroformed as in the above case. |
| Do. | 412 | Large-sized well-nourished pariah. | 10 41 0 | 0 1 35 | 0 8 12 | ... | ... | 10 49 37 | 0 8 0 | Struggled as usual. Artificial respiration was tried 25s. after the breathing had ceased and found to be successful. |
| Nov. 18 | 413 | Full-sized healthy pariah ... ... | 9 41 0 | 0 2 20 | 0 3 50 | ... | ... | 9 45 15 | 0 3 0 | Gradual administration of chloroform on cap. Dog struggled very much. Artificial respiration 25s. after the breathing ceased and found successful. |
| Do. | 414 | Under-sized badly-nourished pariah | 9 50 0 | 0 1 20 | 0 2 30 | ... | ... | 9 53 0 | 0 10 0 | Chloroform administered gradually as above. Artificial respiration by bellows began 30s. after breathing stopped and continued for exactly 10m. After this, needle inserted into heart; no movement. Dog struggled during inhalation. Dog died. |
| Do. | 415 | Full-sized well-nourished pariah... | 10 7 0 | 0 2 3 | 0 2 30 | 0 5 0 | ... | 10 9 45 | 0 6 0 | Chloroformed gradually as above. Artificial respiration by bellows began 15s. after breathing stopped and continued for 6m. No effect. Needle inserted into heart and movements of flag noted. Dog died. Dog struggled as usual during inhalation. |
| Do. | 416 | Full-sized healthy pariah ... ... | 10 20 0 | 0 1 15 | 0 3 10 | ... | ... | 10 23 30 | 0 5 20 | Chloroformed gradually as above. Artificial respiration in the ordinary way began 20s. after respiration ceased and continued for 5m. 20s. Dog died. Struggled a great deal during inhalation. |
| Do. | 417 | Full-grown large-sized pariah ... | 10 32 0 | 0 7 0 | 0 11 18 | ... | ... | 10 43 33 | 0 4 0 | Chloroform given in very small quantities and very gradually with a large admixture of air. Dog revived. Artificial respiration was commenced 15s. after the respiration had ceased. Struggled during administration. |
| Do. | 418 | Do. do. ... | 10 32 30 | 0 4 30 | 0 9 2 | ... | ... | 11 1 57 | 0 3 0 | Chloroformed gradually as in the last case. Artificial respiration was commenced 25s. after the respiration had ceased and proved successful. Struggled during administration. |
| Do. | 419 | Do. do. ... | 11 8 0 | 0 4 12 | 0 7 33 | ... | ... | 11 15 38 | 0 5 0 | Chloroform gradually given as in the last case. Artificial respiration was commenced 25s. after the respiration had ceased. Dog died. Struggled during administration. |
| Do. | 420 | Do. do. ... | 11 21 0 | 0 2 40 | 0 16 2 | ... | ... | 11 37 22 | 0 6 0 | Chloroform gradually given as in the last case. Artificial respiration was commenced 20s. after respiration had stopped and proved successful. |
| Do. | 421 | Full-grown large-sized healthy pariah. | 2 32 0 | 0 5 30 | 0 7 0 | ... | ... | 2 39 20 | 0 8 0 | Chloroform gradually given as in the last case. Artificial respiration commenced 20s. after respiration had stopped. Dog died. Did not struggle much. |
| Do. | 422 | Full-grown under-sized fairly-nourished pariah. | 2 51 30 | 0 1 45 | 0 4 15 | ... | ... | 2 56 0 | 0 1 30 | Chloroformed as in the last case. Artificial respiration commenced 15s. after respiration had ceased. Dog revived. Did not struggle very much. |
| Do. | 423 | Full-grown well-nourished pariah.. | 3 0 0 | 0 2 30 | 0 9 14 | ... | ... | 3 9 34 | 0 3 0 | Chloroform administered in small doses gradually as in the last case. Artificial respiration was commenced 20s. after breathing had ceased and proved successful. Struggled as usual. |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Do. | 424 | Do. do. ... | 3 14 0 | 0 2 8 | 0 18 6 | ... | ... | 3 32 16 | 0 2 0 | Struggled a great deal. Chloroform was administered as in the last case and artificial respiration tried 10s. after breathing had ceased. Dog revived. |
| Do. | 425 | Do. do. ... | 3 36 45 | 0 2 55 | 0 11 50 | ... | ... | 3 49 5 | 0 3 0 | Struggled as usual. Chloroformed as in the previous case. Artificial respiration was commenced 30s. after the breathing had ceased and proved successful. Artificial respiration continued for 3m. |
| Do. | 426 | Do. do. ... | 4 1 0 | 0 2 3 | 0 4 8 | ... | ... | 4 5 38 | 0 3 0 | Struggled. Artificial respiration was commenced 30s. after the breathing had ceased, but did not prove successful. Chloroformed with a large dose and with little air. |
| Nov. 19 | 427 | Full-grown fairly-nourished pariah | 9 48 0 | 0 1 20 | 0 2 5 | ... | ... | 9 50 20 | 0 9 0 | Dog struggled very much; large dose of chloroform given with very little air. Artificial respiration commenced 15s. after breathing had ceased. Gave several gasps after 4m. Artificial respiration unsuccessful. |
| Do. | 428 | Do. do. ... | 10 6 0 | 0 6 15 | 0 13 45 | ... | ... | 10 20 0 | 0 3 30 | Chloroform given in measured doses of 1 drachm at a time. Total given 4 drachms. Artificial respiration commenced 15s. after breathing had ceased and proved successful. The doses of chloroform administered at an interval of 4m. between each dose. Dog struggled slightly. |
| Do. | 429 | Large-sized full-grown well-nourished pariah. | 10 26 0 | 0 3 45 | 0 12 2 | ... | ... | 10 38 7 | 0 2 0 | Chloroform given as in above case. Artificial respiration commenced 5s. after the breathing had ceased. Successful. |
| Do. | 430 | The same dog chloroformed a second time as soon as the cornea became sensitive. | 10 43 4 | 0 2 18 | 0 5 23 | ... | ... | 10 48 32 | 0 2 0 | Chloroformed as in the above case, and artificial respiration commenced 5s. after the breathing had ceased. Successful. |
| Do. | 431 | The same dog chloroformed for the third time as soon as the cornea became sensitive. | 10 55 0 | 0 1 43 | 0 10 3 | ... | ... | 11 5 8 | 0 4 0 | Chloroformed as in the last case. Artificial respiration was commenced 5s. after the breathing had ceased, but proved unsuccessful. |
| Do. | 432 | Young monkey ... ... ... | 11 13 0 | 0 0 58 | 0 2 0 | ... | ... | 11 15 5 | 0 6 0 | Chloroformed as in the last case. Artificial respiration was commenced 5s. after the breathing had ceased, but proved unsuccessful. The bellows were used in this case for artificial respiration and found unsuitable. |
| Do. | 433 | Do. ... ... ... | 11 33 0 | 0 1 25 | 0 5 0 | ... | ... | 11 38 5 | 0 4 0 | Chloroformed as in the last case. Artificial respiration was commenced 5s. after the breathing had ceased and proved successful. Artificial respiration was carried on in the ordinary manner with the hands. |
| Do. | 434 | Do. ... ... ... | 12 0 0 | 0 1 15 | 0 4 56 | ... | ... | 12 5 6 | 0 3 0 | Artificial respiration was commenced 10s. after the respiration had ceased and was unsuccessful. |
| Nov. 20 | 435 | Do. ... ... ... | 9 26 0 | 0 8 0 | 0 17 20 | ... | ... | 9 43 50 | 0 3 0 | Chloroform administered in 1 dr. doses at intervals of 5 minutes with plenty of air. Artificial respiration commenced 30s. after the breathing had ceased. Animal revived. Total chloroform administered 3 drs. |
| Do. | 436 | Do. ... ... ... | 10 23 0 | 0 1 15 | 0 1 58 | ... | ... | 10 25 28 | 0 2 0 | Chloroform administered as in the last case. Artificial respiration commenced 30s. after the respiration had ceased. Proved successful. |

**VI (a).**—*Animals chloroformed for one hour, allowed to revive, and killed with chloroform the next day.*

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Do. | 437 | Full-grown pariah dog ... ... | 10 5 0 | ... | ... | ... | ... | ... | ... | Chloroformed for an hour and allowed to revive for a further observation the next day. |
| | | November 21st ... ... ... | 10 28 0 | 0 0 55 | 0 2 4 | 0 2 35 | 0 4 55 | ... | ... | 21st—Post-mortem made after chloroforming the animal to death. Weight 24 lbs. Liver and portal system congested generally; kidneys and spleen congested. Heart—left side distended with arterial blood, and right side venous. Lungs and trachea were normal. |
| Do. | 438 | Young monkey ... ... ... | 10 8 0 | ... | ... | ... | ... | ... | ... | Do. do. |
| | | November 21st ... ... ... | 11 19 0 | 0 0 58 | 0 1 45 | 0 2 40 | 0 4 10 | ... | ... | Weight 5 lbs. Post-mortem made after chloroforming to death. Post-mortem appearances as in 437. |

| 1<br>Date. | 2<br>No. | 3<br>Description of dog. | 4<br>Time at which inhalation commenced. | 5<br>Cornea became insensible after | 6<br>Respiration ceased after | 7<br>Pulse stopped after | 8<br>Heart ceased beating after | 9<br>Artificial respiration commenced at | 10<br>Artificial respiration continued for | 11<br>Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|
| 1889. | | | h. m. s. | h. m. s. | h. m. s. | h. m. s. | h. m. s. | h. m. s. | h. m. s. | |
| Nov. 20 | 439 | Full-grown pariah dog ... ... | 10 0 0 | ... | ... | ... | ... | ... | ... | Chloroformed for an hour and allowed to revive for a further observation the next day. |
| | | November 21st ... ... ... | 9 57 0 | 0 1 0 | 0 2 5 | 0 2 45 | 0 5 55 | ... | ... | 21st—Chloroformed to death and a post-mortem made. Weight 26 lbs. The same post-mortem appearances were noticed as in the last case. |
| Do. | 440 | Young monkey ... ... ... | 10 0 0 | ... | ... | ... | ... | ... | ... | Do. do. |
| | | November 21st ... ... ... | 11 5 0 | 0 1 0 | 0 2 3 | 0 2 15 | 0 4 35 | ... | ... | 21st—Chloroformed to death, and a post-mortem made. The same post-mortem conditions were noticed as in the last case. Weight 8 lbs. |
| Do. | 441 | Young Monkey ... ... ... | 10 0 0 | ... | .. | ... | ... | ... | ... | Do. do. |
| | | November 21st ... ... ... | 10 34 0 | 0 1 0 | 0 2 0 | 0 4 0 | 0 6 15 | ... | ... | 21st—Chloroformed to death, and a post-mortem made. No difference to be seen in the post-mortem appearance. Weight 8 lbs. |
| Do. | 442 | Full-grown large pariah dog ... | 10 0 0 | ... | ... | ... | ... | ... | ... | Do. do. |
| | | November 21st ... ... ... | 10 0 0 | 0 0 50 | 0 1 30 | 0 2 5? | 0 6 0 | ... | ... | 21st—Chloroformed to death, and a post-mortem made. Weight 80 lbs. Post-mortem appearance as in the other cases. |
| ***V (a).—Artificial respiration tried in this case.*** | | | | | | | | | | |
| Do. | 443 | Young monkey ... ... ... | 10 36 0 | 0 2 50 | 0 8 2 | ... | ... | 10 44 32 | 0 2 0 | Chloroformed as in Experiment 436 and artificial respiration commenced 30s. after the breathing had ceased. Monkey revived. |
| ***I (c).—Monkeys kept fasting for 24 hours and chloroformed to death with large doses on cloth cap inhaler.*** | | | | | | | | | | |
| Do. | 444 | Young monkey ... ... ... | 10 50 20 | 0 2 1 | 0 6 13 | 0 7 0 | 0 10 24 | ... | ... | The monkey was fasting for 24 hours. Did not struggle. |
| Do. | 445 | Do. . ... ... | 11 5 0 | 0 1 56 | 0 3 18 | 0 4 2 | 0 8 16 | ... | ... | Do. do. |
| Do. | 446 | Do. ... ... ... | 11 14 0 | 0 0 58 | 0 2 2 | 0 3 5 | 0 4 6 | ... | ... | In this case the effect of 1 oz. of chloroform on the cap was tried. Struggled. |
| Do. | 447 | Do. ... ... ... | 11 20 0 | 0 1 6 | 0 1 45 | 0 2 0 | 0 8 40 | ... | ... | Do. do. |
| ***V (a).—Artificial respiration tried in these cases.*** | | | | | | | | | | |
| Do. | 448 | Young monkey ... ... ... | 11 29 0 | 0 2 6 | 0 3 20 | ... | ... | 11 32 50 | 0 2 0 | Chloroform administered in 1 dr doses every 5m. on cloth cap as in case 443. Artificial respiration was commenced 30s. after the breathing had ceased and proved successful. Did not struggle. |
| Do. | 449 | Young monkey was partially choked when brought on table and had to be revived. | 11 47 0 | 0 1 3 | 0 1 52 | ... | .. | 11 49 22 | 0 1 30 | Chloroformed as in the previous case. Artificial respiration was commenced 30s. after the breathing had ceased and proved successful. Did not struggle. |
| Do. | 450 | Young monkey ... ... ... | 11 53 0 | 0 2 0 | 0 4 14 | ... | ... | 11 57 49 | 0 2 1 | Chloroformed as in Experiment 449. Did not struggle. Artificial respiration was tried 35s. after the breathing had ceased and proved successful. |
| Do. | 451 | Full-grown monkey ... ... | 3 11 0 | 0 1 50 | 0 5 23 | ... | ... | 3 17 3 | 0 4 0 | Struggled a great deal. Chloroformed as in Experiment 450. Artificial respiration was commenced 40s. after the respiration had ceased and proved successful. |
| Do. | 452 | Do. ... ... | 3 30 0 | 0 1 17 | 0 6 0 | ... | ... | 3 36 45 | 0 3 0 | Struggled. Chloroform given as in Experiment 451 and artificial respiration 45s. after the respiration had ceased. Monkey revived. |

### VI (b.)—*Animals kept under the influence of ether for one hour and chloroformed to death the next day.*

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Do. | 453 | Large-sized well-nourished dog ... | 3 0 0 | ...... | ...... | ...... | ...... | ...... | ...... | Large doses of ether administered for one hour and allowed to recover for further observation. |
| | | November 21st ... ... ... | 10 17 10 | 0 1 3 | 0 1 40 | 0 2 5 | 0 9 30 | ...... | ...... | 21st—Chloroformed to death by large doses of chloroform. Gasped 21 times 4m. 30s. after respiration ceased. Did not struggle. Weight 30 lbs. Post-mortem appearances—congestion of liver and portal system generally. Kidneys highly congested. The spleen was three times larger than normal and puckered. Trachea and lungs normal. Heart—right side distended with venous blood and left with arterial. The post-mortem appearances corresponded generally with those seen in the animals that had had chloroform the day before instead of ether. *Vide* VI (a.) |
| Do. | 454 | Large-sized well-nourished dog ... | 3 0 0 | ...... | ...... | ...... | ...... | ...... | ...... | Large doses of ether administered for one hour and allowed to recover for further observation. |
| | | November 21st ... ... ... | 10 42 0 | 0 1 10 | 0 2 18 | 0 2 40 | 0 6 15 | ...... | ...... | 21st—Chloroformed in the same manner as in the above case. Dog struggled violently. Weight 32 lbs. Post-mortem appearances as in the last case with the exception of the spleen, which was simply congested and not enlarged. |
| Do. | 455 | Large-sized well-nourished dog ... | 3 0 0 | ...... | ...... | ...... | ...... | ...... | ...... | Large dose of ether administered for one hour and allowed to recover for further observation. |
| | | November 21st ... ... ... | 10 50 0 | 0 1 15 | 0 1 50 | 0 2 10 | 0 3 10 | ...... | ...... | 21st—Chloroformed to death in the same manner as in the last case. the next day. Dog struggled violently. Post-mortem appearances as in the last case. Weight 33 lbs. |
| Do. | 456 | Full-grown small-sized monkey ... | 3 0 0 | ...... | ...... | ...... | ...... | ...... | ...... | Large dose of ether administered for one hour, and allowed to recover for further observation. |
| | | November 21st ... ... ... | 11 27 0 | 0 1 10 | 0 3 25 | 0 3 43 | 0 5 30 | ...... | ...... | 21st—Chloroformed in same manner as in above case. Struggled a little. Weight 8 lbs. Post-mortem appearances as in the last case. |
| Do. | 457 | Small monkey ... ... ... | 3 0 0 | ...... | ...... | ...... | ...... | ...... | ...... | Large dose of ether administered for one hour and allowed to recover for further observation. |
| | | November 21st ... ... ... | 10 57 0 | 0 1 10 | 0 3 45 | 0 4 30 | 0 4 45 | ...... | ...... | 21st—Chloroformed as in the above case. Did not struggle at all. Weight 8 lbs. Post-mortem appearances as in the last case. |
| Do. | 458 | Small monkey ... ... ... | 3 0 0 | ...... | ...... | ...... | ...... | ...... | ...... | Large dose of ether administered for one hour and allowed to recover for further observation. |
| | | November 21st ... ... ... | 11 40 30 | 0 1 0 | 0 3 0 | 0 3 30 | 0 4 15 | ...... | ...... | 21st—Chloroformed as above. Weight 5 lbs. Post-mortem appearances as in the last case. |

### V (a.)—*Artificial respiration tried in these cases.*

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Nov. 22 | 459 | Medium-sized monkey ... ... | 10 0 0 | 0 2 0 | 0 12 25 | ... | ... | 10 13 5 | 0 1 0 | Chloroform administered in small doses with plenty of air. Artificial respiration commenced 40s. after respiration had ceased and proved successful. Weight 7 lbs. |
| Do. | 460 | Small monkey ... ... ... | 10 17 0 | 0 0 55 | 0 1 55 | ... | ... | 10 19 35 | 0 13 0 | Chloroformed as in the above case but with less air. Artificial respiration commenced 40s. after respiration had ceased. Unsuccessful. |
| Do. | 461 | Medium-sized monkey ... ... | 10 25 0 | 0 2 0 | 0 6 25 | ... | ... | 10 32 10 | 0 1 40 | Chloroformed as in the above case. Artificial respiration commenced 45s. after respiration had ceased and proved successful. |
| Do. | 462 | Medium-sized monkey, subject of Experiment 274. | 10 39 0 | 0 1 5 | 0 2 15 | ... | ... | 10 42 5 | 0 16 0 | Chloroformed as in above case. Artificial respiration commenced 50s. after respiration had ceased. Proved successful. Weight 7 lbs. |
| Do. | 463 | Full grown monkey ... ... | 12 0 0 | 0 2 4 | 0 8 13 | ... | ... | 12 9 8 | 0 10 0 | Chloroformed as in the above case and artificial respiration commenced 55s. after the breathing had ceased. Artificial respiration unsuccessful. The monkey gasped four times before he died. |

| 1<br>Date. | 2<br>No. | 3<br>Description of dog. | 4<br>Time at which inhalation commenced. | 5<br>Cornea became insensible after | 6<br>Respiration ceased after | 7<br>Pulse stopped after | 8<br>Heart ceased beating after | 9<br>Artificial respiration commenced at | 10<br>Artificial respiration continued for | 11<br>Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | h. m. s. | h. m. s. | h. m. s. | h. m. s. | h. m. s. | h. m. s. | h. m. s. | |
| Nov. 22 | 464 | Full-grown monkey... ... ... | 11 20 0 | 0 1 40 | 0 9 27 | ... | ... | 11 30 22 | 0 7 0 | Chloroformed as in the above case, and artificial respiration commenced 55s. after the breathing had ceased. Unsuccessful. |
| Do. | 465 | Do. ... ... ... | 11 38 0 | 0 0 53 | 0 3 0 | ... | ... | 11 41 55 | 0 1 0 | Chloroformed as in the above case, and artificial respiration commenced 55s. after the breathing had ceased. Monkey revived. |
| Do. | 466 | Subject of a former experiment ... | 11 51 0 | 0 1 3 | 0 3 16 | ... | ... | 11 55 11 | 0 2 0 | Chloroformed as in the above case, and artificial respiration tried 55s. after the respiration had ceased. Proved successful. |

**VII.**—*Administration of a definite quantity of chloroform.*

| 1<br>Date. | 2<br>No. | 3<br>Description of dog. | 4<br>Time at which inhalation commenced. | 5<br>Cornea became insensible after | 6<br>Respiration ceased after | 7<br>Pulse stopped after | 8<br>Heart ceased beating after | 9<br>Artificial respiration commenced at | 10<br>Artificial respiration continued for | 11<br>Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|
| Do. | 467 | Full-grown middle-sized pariah ... | 2 23 0 | ... | ... | ... | ... | ... | ... | Chloroformed in a box at 2h. 23m. Fell down at 2h. 30s. Taken out and placed on the table at 2h. 31m. Trachea opened and tube inserted at 2h. 40m. A definite quantity of chloroform and air was then administered through the bellows from a box 8 cubic feet in capacity and into which two ounces of chloroform had been placed at 2h. 45m. At 3h. 45m. the dog was found to be coming out and another ounce was placed in the box. Needle placed in heart at 4h. Heart stopped at 4h. 8m. 13s. |
| Nov. 23 | 468 | Full-grown well-nourished pariah.. | 9 50 0 | ... | ... | ... | 0 40 0 | ... | ... | Chloroformed in a dealwood box at 9h. 30m. Fell down at 9h. 40m. Was taken out of the box at 9h. 45m. Trachea opened and tube introduced at 9h. 50m. Chloroform given as in the last case with bellows and from the tin box. In this case the heart ceased beating judging from needle in thorax at 10h. 20s. and commenced again after a full minute and then ceased finally at 10h. 30m. The time of cessation of respiration could not be noted in this nor in the last case on account of the bellows being used until death occurred. |

**V. (a).**—*Artificial Respiration tried in these cases.*

| 1<br>Date. | 2<br>No. | 3<br>Description of dog. | 4<br>Time at which inhalation commenced. | 5<br>Cornea became insensible after | 6<br>Respiration ceased after | 7<br>Pulse stopped after | 8<br>Heart ceased beating after | 9<br>Artificial respiration commenced at | 10<br>Artificial respiration continued for | 11<br>Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|
| Do. | 469 | Full-grown monkey... ... ... | 10 56 0 | 0 2 5 | 0 4 18 | 0 4 30 | ... | 11 1 13 | 0 2 0 | Chloroformed in large doses with cloth inhaler. Artificial respiration was commenced 55s. after the respiration had ceased and proved successful. Weight 6 lbs. |
| Do. | 470 | Do. ... ... ... | 11 5 0 | 0 1 25 | 0 3 40 | 0 4 1 | ... | 11 9 40 | 0 2 30 | Chloroformed as in the last experiment, and artificial respiration commenced one minute after the respiration had ceased. The monkey revived. |
| Do. | 471 | Do. Subject of Experiment 469. | 11 18 18 | 0 2 0 | 0 4 53 | Not noted. | ... | 11 24 11 | 0 6 0 | Chloroformed as in the above experiment, and artificial respiration commenced one minute after the breathing had ceased. Needle in heart stopped vibrating at 11h. 30m. Unsuccessful |
| Do. | 472 | Young monkey ... ... ... | 11 31 0 | 0 1 48 | 0 6 2 | Do. | ... | 11 38 9 | 0 6 0 | Chloroformed as in the above experiment. The respiration in this case ceased after 3m. 32s. for 40s. and commenced again. More chloroform had to be given and he ceased breathing for the second time after 1m. 2s. from time of inhalation. Artificial respiration was commenced 1m. 7s. after the breathing had ceased, and proved unsuccessful. Needle in heart at 11h. 42m. found to be vibrating. |
| Do. | 473 | Do. ... ... ... | 11 46 0 | 0 1 35 | 0 3 23 | ... | ... | 11 50 23 | 0 2 0 | Chloroformed as in the above experiment. Artificial respiration was commenced one minute after the breathing had ceased and proved successful. Struggled. |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Do. | 473 | Young Monkey ... ... ... | 11 46 0 | 0 1 35 | 0 3 23 | ... | ... | 11 50 23 | 0 2 0 | Chloroformed as in the above experiment. Artificial respiration was commenced one minute after the breathing had ceased, and proved successful. Struggled. |

V (b).—*Artificial respiration tried on dogs poisoned with phosphorus.*

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Nov. 25 | 474 | Full-grown large pariah; has had ¾ grain of phosphorus a day since 22nd instant. | 10 18 0 | 0 1 0 | 0 2 53 | ... | ... | 10 21 33 | 0 6 0 | Chloroformed in large doses with inhaler tightly held over face. Struggled a great deal. Artificial respiration was commenced one minute after the breathing had ceased and proved unsuccessful. Weight 30 lbs. The dog gasped several times after artificial respiration had been practised for a minute.<br>Post-mortem appearances—Liver found ruptured in three places, and the peritoneal cavity full of dark blood. Liver distinctly fatty (mottled), soft and friable. Heart soft, mottled on surface. Endocardium pale, lung dry and non-cripitant. Lines of medullary rays in kidneys were well marked. |
| Do. | 475 | Full-grown large-sized pariah; has had ¾ grain of phosphorus a day since 22nd instant. | 10 43 0 | 0 1 48 | 0 2 18 | ... | ... | 10 46 18 | 0 2 0 | Chloroformed as in the above experiment and artificial respiration commenced one minute after the breathing had ceased. The dog was revived after artificial respiration had been practised two minutes. Struggled during inhalation. |
| Do. | 476 | Full-grown pariah; has had phosphorus as in the above case. | 10 5 20 | 0 2 30 | 0 3 0 | ... | ... | 10 56 0 | 0 6 0 | Chloroformed as in the last case, and artificial respiration commenced one minute after the breathing had ceased. Dog died. The needle in heart was found to be vibrating until 11h. 3m. 12s. Struggled during inhalation. Weight 30 lbs.<br>Post-mortem appearances same as No. 475, with the exception that the liver was not ruptured. |
| Do. | 477 | Do. do. ... | 11 8 0 | 0 1 52 | 0 2 52 | ... | ... | 11 11 52 | 0 7 0 | Chloroformed as in the last experiment. Artificial respiration was tried 1 minute after the breathing had ceased and proved unsuccessful. Weight 28 lbs. Heart acted for 5 minutes after the respiration had ceased. Struggled during inhalation.<br>Post-mortem appearances as in No. 476. |
| Do. | 478 | Full-grown large pariah; was given phosphorus as in the previous cases. | 11 18 0 | 0 2 1 | 0 4 1 | ... | ... | 11 22 45 | 0 6 0 | Chloroformed as in above case, and artificial respiration tried unsuccessfully 45s. after the breathing had ceased. Struggled a great deal.<br>Post-mortem appearances as in No. 477. |
| Do. | 479 | Full-grown large pariah; has had phosphorus as in the last case. | 11 32 0 | 0 2 33 | 0 3 15 | ... | ... | 11 36 0 | 0 13 0 | Chloroformed as in the above case. Artificial respiration was tried 45s. after breathing had ceased. Needle thrust into the heart at 11h. 47m.; was seen to be vibrating. Dog died at 11h. 50m. 5s. Weight 22 lbs.<br>Post-mortem appearances as in No. 478. |
| Do. | 480 | Full-grown large pariah; has had the same amount of phosphorus as in the previous cases. | 11 51 0 | 0 1 53 | 0 2 13 | ... | ... | 11 53 51 | 0 3 0 | Chloroformed as in the above cases and artificial respiration tried 38s. after the breathing had ceased. The dog revived 2 minutes after artificial respiration had been commenced. |
| Do. | 481 | Full-grown large pariah; has had phosphorus as in the previous cases. | 12 0 0 | 0 1 3 | 0 2 18 | ... | ... | 12 2 53 | 0 6 0 | Chloroformed as in No. 480. Artificial respiration commenced 35s. after breathing had ceased. Dog died. Weight 35 lbs.<br>Post-mortem appearances as in No. 479. |

V (a).—*Artificial Respiration tried in these cases without the previous administration of phosphorus.*

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Do. | 482 | Young monkey .. ... ... | 3 29 0 | 0 1 13 | 0 3 56 | ... | ... | 3 33 26 | 0 3 0 | Chloroformed in large doses with cloth inhaler tightly held over the face. Artificial respiration was commenced 30s. after the breathing had ceased and proved unsuccessful. |
| Do. | 483 | Do ... ... ... | 3 39 0 | 0 0 56 | 0 4 2 | ... | ... | 3 43 32 | 0 6 0 | Ditto. Artificial respiration was commenced 30s. after the breathing had ceased and proved unsuccessful. |
| Do. | 484 | Do. ... ... ... | 3 52 0 | 0 1 0 | 0 3 39 | ... | ... | 3 56 9 | 0 15 0 | Ditto. Artificial respiration was commenced 30s. after the breathing had ceased and continued 15m. Proved unsuccessful. |
| Do. | 485 | Do. ... ... ... | 4 8 0 | 0 1 45 | 0 4 0 | ... | ... | 4 12 30 | 0 1 0 | Ditto. Artificial respiration was commenced 30s. after the breathing had ceased and proved successful after a minute. |

| 1<br>Date. | 2<br>No. | 3<br>Description of dog. | 4<br>Time at which inhalation commenced. | 5<br>Cornea became insensible after | 6<br>Respiration ceased after | 7<br>Pulse stopped after | 8<br>Heart ceased beating after | 9<br>Artificial respiration commenced at | 10<br>Artificial respiration continued for | 11<br>Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|
| | | | h. m. s. | h. m. s. | h. m. s. | h. m. s. | h. m. s. | h. m. s. | h. m. s. | |
| **V (c).**—*The abdomen was opened in these cases and aromatic spirits of ammonia injected into the stomach before artificial respiration was tried.* | | | | | | | | | | |
| Nov. 26 | 486 | Full-sized healthy pariah ... ... | 9 47 30 | 0 1 0 | ... | ... | ... | ... | ... | Operation of opening abdomen began at 9h. 50m. Cornea sensitive at 9h. 54m. More chloroform at 9h. 54m. 10s. Cornea insensitive and chloroform removed at 9h. 55m. 30s. 1 dr. spirits ammonia co. being injected into stomach by hypodermic syringe at 9h. 58m. 25s. Injection completed at 9h. 59m. 10s. Pushed chloroform at 10h. 4m. Dog being still under. Respiration ceased at 10h. 7m. 50s. Artificial respiration commenced 10h. 8m. 20s., 30s. after breathing ceased, and continued for 5m. 40s. and proved unsuccessful. Weight 34 lbs. |
| Do. | 487 | Do. do. ... ... | 10 17 0 | ... | ... | ... | ... | ... | ... | Chloroform inhalation begun at 10h. 17m. Cornea insensitive 10h. 18m. 10s. Respiration ceased suddenly 10h. 19m., and artificial respiration commenced immediately and continued for 4m. Operation of opening abdomen begun at 10h. 24m. Injection of 1 dr. spirits of ammonia co. begun at 10h. 26m. 30s. into stomach as in above case. Finished at 10h. 27m. Pushed chloroform at 10h. 30m., dog being still under. Respiration ceased at 10h. 36m. 40s. Artificial respiration commenced at 10h. 37m. 0s. after breathing had ceased and continued for 2m. Dog revived. Weight 29 lbs. |
| **V (a).**—*Artificial respiration tried.* | | | | | | | | | | |
| Do. | 488 | The same dog chloroformed again. | 10 46 0 | 0 0 53 | 0 3 10 | ... | ... | 10 49 40 | 0 2 0 | Chloroformed with large doses and artificial respiration commenced 30s. after the breathing had ceased. Unsuccessful. |
| Do. | 489 | Young monkey, weighed 6 lbs. ... | 11 1 0 | 0 0 58 | 0 2 16 | ... | ... | 11 3 46 | 0 4 0 | Chloroformed as in the above experiment. Artificial respiration was commenced 30s. after the respiration had ceased and proved successful. |
| **X (k).**—*Monkeys chloroformed to death after the subcutaneous injection of aromatic spirits and ammonia.* | | | | | | | | | | |
| Do. | 490 | Young monkey ; had 20 minims of spirits ammonia aromatic injected under the skin at 11h. 2m. | 11 11 0 | 0 1 18 | 0 2 16 | 0 2 23 | 0 9 23 | ... | ... | Chloroform in large doses till death occurred. Weight 6 lbs. gasped twice before the heart stopped. |
| Do. | 491 | Young monkey ; had 20 minims of spirits ammonia co. injected under the skin at 11h. 12m. | 11 21 0 | 0 0 48 | 0 1 59 | 0 2 6 | 0 7 0 | ... | ... | Idem. Weight 8 lbs. |
| Do. | 492 | Full-grown monkey; had 20 minims of spirits of ammonia aromatic injected at 11h. 20m. 0s. | 11 28 0 | 0 1 18 | 0 2 53 | Not noted. | 0 10 12 | ... | ... | Do. Weight 12 lbs. |
| Do. | 493 | Full-grown monkey; had 20 minims of spirits of ammonia aromatic at 11h. 25m. | 11 35 0 | 0 1 12 | 0 2 27 | 0 2 48 | 0 15 0 | ... | ... | Do. do. |
| Do. | 494 | Full-grown monkey, small-sized : had 20 minims spirits of ammonia co. at 2h. 45m. | 2 50 0 | 0 1 0 | 0 1 30 | 0 2 10 | 0 4 0 | ... | ... | Do. Weight 5 lbs. |
| Do. | 495 | Young monkey ; had 20 minims spirits of ammonia co. at 2h. 55m. | 3 0 0 | 0 1 0 | 0 2 10 | 0 3 30 | 0 5 50 | ... | ... | Do. Respiration returned 35s. after it ceased and continued for 1m., after which it ceased entirely. |

**V (a)**—*Artificial respiration tried.*

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Do. | 496 | Young monkey ... ... ... | 3 14 0 | 0 0 56 | 0 1 58 | ... | ... | 3 16 28 | 0 5 0 | Chloroformed by 1 dr. doses at a time, the monkey being in the erect position. Artificial respiration was commenced 30s. after the breathing had ceased, the monkey having been inverted before it was begun. Unsuccessful. The heart stopped beating 7 minutes after inhalation. Weight 6 lbs. |
| Do. | 497 | Do. ... ... ... | 3 30 0 | 0 1 5 | 0 2 11 | ... | ... | 3 32 41 | 0 14 0 | Ditto. Artificial respiration commenced 30s. after the breathing had ceased. The monkey, being inverted, proved unsuccessful. A needle in the heart was found to be vibrating 15m. after inhalation. |
| Do. | 498 | Do. ... ... ... | 3 57 0 | 1 4 0 | 0 3 0 | ... | ... | 4 0 30 | 0 4 0 | Ditto. Artificial respiration was commenced 30s. after the breathing had ceased, the monkey having been inverted, and proved successful. |

**I (m)**—*Monkeys chloroformed to death in the erect position and with large doses on cloth inhaler.*

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Do. | 499 | Full-grown monkey ... ... | 10 37 0 | 0 1 30 | 0 5 0 | 0 9 30 | 0 11 15 | ... | ... | Chloroformed in the erect position and the anæsthetic pushed until death occurred. |
| Do. | 500 | Small monkey ... ... ... | 10 55 0 | 0 1 20 | 0 3 30 | 0 5 0 | 0 6 0 | ... | ... | Do. do. Weight 5 lbs. |
| Do. | 501 | Do. ... ... ... | 11 5 0 | 0 1 0 | 0 2 46 | 0 3 5 | 0 4 6 | ... | ... | Do. do. do. |
| Do. | 502 | Do. ... ... ... | 11 13 30 | 0 1 5 | 0 1 50 | 0 3 15 | 0 3 38 | ... | ... | Do. The thorax was opened in this case and the heart seen to have stopped immediately after the needle in that organ ceased to vibrate. Weight 5 lbs. |
| Do | 503 | Young monkey ... ... ... | 11 27 0 | 0 0 52 | 0 4 18 | 0 4 23 | 0 4 40 | ... | ... | Do. do. Weight 8 lbs. |
| Do | 504 | Do. ... ... ... | 11 43 30 | 0 1 0 | 0 6 2 | 0 6 23 | 0 10 12 | ... | ... | Do. do. Weight 6 lbs. |

**V (a)**—*Artificial respiration tried in these cases.*

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Do. | 505 | Do. ... ... ... | 11 57 0 | 0 1 5 | 0 5 16 | ... | ... | 12 2 46 | 0 5 0 | Chloroformed in the recumbent position with large doses and inverted before artificial respiration was begun. Artificial respiration was commenced 30s. after the breathing had ceased and proved ineffectual. Weight 7 lbs. |
| Nov. 27. | 506 | Do. ... ... ... | 2 51 0 | 0 1 15 | 0 5 0 | ... | ... | 2 56 30 | 0 2 0 | Chloroformed as above. Proved unsuccessful. Artificial respiration commenced after 30s. and continued for 2m. |
| Do. | 507 | Full-grown monkey ... ... | 3 7 0 | 0 1 45 | 0 5 15 | ... | ... | 3 13 0 | 0 7 0 | Chloroformed as above. Artificial respiration commenced after 45s. and continued for 7m. unsuccessfully. |
| Do. | 508 | Young monkey ... ... ... | 3 20 0 | 0 0 54 | 0 5 0 | ... | ... | 3 28 45 | 0 4 0 | Chloroformed as in the above case and artificial respiration commenced 45s. after the breathing had ceased. Successful. |
| Do. | 509 | Do. ... ... ... | 3 32 0 | 2 44 0 | 0 6 2 | ... | ... | 3 38 22 | 0 3 0 | Chloroformed as in the above case. Struggled a great deal. Artificial respiration was commenced 20s. after the breathing had ceased and proved successful. Weight 8 lbs. |
| Do. | 510 | The same monkey after he had revived. | 3 44 0 | 0 0 42 | 0 1 14 | ... | .. | 3 45 44 | 0 6 0 | Chloroformed as in the above case. Artificial respiration was commenced 30s. after the breathing had ceased. Unsuccessful. |
| Do. | 511 | Full-grown monkey ... ... | 3 55 0 | 0 2 18 | 0 5 16 | ... | ... | 4 1 16 | 0 2 0 | Chloroformed as in the last experiment. Artificial respiration was commenced one minute after the breathing had ceased and found unsuccessful. |

**V (b)**.—*Artificial respiration tried on dogs sick from phosphorus poisoning.*

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Nov. 30. | 512 | Dog sick from phosphorus poisoning; had 1 gr. of phosphorus daily for 3 days from 25th instant. | 10 23 0 | 0 1 40 | 0 4 15 | ... | ... | 10 27 40 | 0 2 0 | Struggled. Artificial respiration was commenced 25s. after the breathing had ceased and succeeded in reviving the dog. |
| Do. | 513 | Do. do. ... | 10 30 0 | 0 1 16 | 0 2 4 | ... | ... | 10 32 44 | 0 1 30 | Struggled. Artificial respiration was commenced 40s. after the breathing had ceased and proved successful. |
| Do. | 514 | Do. do. ... | 10 47 0 | 0 1 10 | 0 2 14 | ... | ... | 10 50 4 | 0 7 0 | Struggled a great deal. Artificial respiration was commenced 50m. after breathing had ceased. Dog died. Post-mortem appearances—Liver ruptured. It was soft, friable and mottled and distinctly fatty. Heart paler than usual. |

| 1<br>Date. | 2<br>No. | 3<br>Description of dog. | 4<br>Time at which inhalation commenced. | 5<br>Cornea became insensible after | 6<br>Respiration ceased after | 7<br>Pulse stopped after | 8<br>Heart ceased beating after | 9<br>Artificial respiration commenced at | 10<br>Artificial respiration continued for | 11<br>Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|
| 1889. | | | h. m. s. | h. m. s. | h. m. s. | h. m. s. | h. m. s. | h. m. s. | h. m. s. | |

**V (e).**—*Three dogs sick from phosphorus poisoning etherised and artificial respiration tried on them.*

| Date. | No. | Description of dog. | 4 | 5 | 6 | 7 | 8 | 9 | 10 | Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|
| Nov. 30 | 515 | Dog sick from phosphorus poisoning; had 1 gr. of phosphorus daily for 3 days from 25th instant. | 11 3 0 | 0 2 16 | 0 6 18 | ... | ... | 11 9 43 | 0 6 0 | Struggled. Artificial respiration was commenced 25s. after the respiration had ceased and proved unsuccessful. |
| Do. | 516 | Do. do. ... | 11 35 0 | 0 1 45 | 0 12 10 | ... | ... | 11 47 40 | 0 2 0 | Dog struggled much. Artificial respiration commenced 30s. after respiration ceased and continued for 2m. Proved successful. |
| Do. | 517 | Do. do. ... | 3 44 0 | 0 1 25 | 0 8 3 | ... | ... | 3 52 53 | 0 6 0 | Artificial respiration was commenced 50s. after the respiration had ceased. Dog died. |

**III (g).**—*12 Dogs injected with cocaine into the peritoneum about 10 minutes before being chloroformed to death with large doses on cloth inhaler.*

| Date. | No. | Description of dog. | 4 | 5 | 6 | 7 | 8 | 9 | 10 | Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|
| Dec. 2 | 518 | Full-grown well-nourished pariah. | 10 40 0 | 0 1 10 | 0 2 10 | 0 4 5 | 0 6 10 | ... | ... | Struggled. Cocaine ¼ gr. injected at 10h. 30m. |
| Do. | 519 | Do. do. .. | 11 1 0 | 0 1 26 | 0 2 18 | 0 4 20 | 0 8 13 | ... | ... | Struggled very much. Cocaine ¼ gr. injected at 10h. 30m. |
| Do. | 520 | Full-grown thin pariah ... ... | 11 11 0 | 0 1 11 | 0 2 30 | 0 3 24 | 0 8 0 | ... | ... | Do. Cocaine ½ gr. injected at 11h. |
| Do. | 521 | Small-sized full-grown pariah ... | 11 29 0 | 0 2 0 | 0 2 40 | 0 2 50 | 0 6 10 | ... | ... | Did not struggle. Injected with ¼ gr. of cocaine at 11h. 20m. |
| Do. | 522 | Full-grown lean pariah ... ... | 11 43 30 | 0 1 0 | 0 3 56 | 0 5 18 | 0 9 2 | ... | ... | Struggled. Cocaine 1 gr. injected at 11h. 35m. |
| Do. | 523 | Emaciated full-grown pariah ... | 11 55 0 | 0 1 5 | 0 6 24 | 0 6 28 | 0 10 38 | ... | ... | Do. Cocaine 1 gr. injected at 11h. 45m. |
| Dec. 3 | 524 | Under-sized ill-nourished pariah ... | 10 0 0 | 0 0 52 | 0 1 50 | 0 2 12 | 0 5 20 | ... | ... | Cocaine gr. 1½ injected at 9h. 45m. inhalation. Dog struggled. |
| Do. | 525 | Full-sized well-nourished pariah ... | 10 9 0 | 0 1 5 | 0 1 35 | 0 1 50 | 0 5 58 | ... | ... | Cocaine gr. 1½ injected at 9h. 58m. Dog struggled. |
| Do. | 526 | Full-grown powerful pariah ... | 10 33 0 | 0 0 50 | 0 1 40 | 0 1 58 | 0 6 5 | ... | ... | Cocaine gr. 1½ injected at 10h. 23m. Gave great trouble in being chloroformed. Struggled a great deal and had to be held down with much force. |
| Do. | 527 | Full-grown middle-sized pariah ... | 3 57 30 | 0 1 40 | 0 4 30 | 0 5 0 | 0 8 28 | ... | ... | Cocaine gr. 2 injected at 2h. 43m. Dog struggled. |
| Do. | 528 | Full-grown well-nourished pariah. | 3 9 0 | 0 0 48 | 0 2 0 | 0 2 23 | 0 8 13 | ... | ... | Cocaine gr. 2 injected at 2h. 54m. Did not struggle. |
| Do. | 529 | Full-grown small-sized pariah ... | 3 24 0 | 0 1 14 | 0 2 18 | 0 3 6 | 0 5 33 | ... | ... | Cocaine gr. 2 injected at 3h. 6m. Shortly after the injection the dog became excited. He was then seized with convulsions; fell down at 3h. 15m. Did not struggle during the experiment. |

**III (h).**—*Twelve dogs injected with strychnine before being chloroformed to death with large doses on cloth inhaler.*

| Date. | No. | Description of dog. | 4 | 5 | 6 | 7 | 8 | 9 | 10 | Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|
| Do. | 530 | Full-grown well-nourished pariah. | 3 39 0 | 0 1 2 | 0 1 40 | 0 2 5 | 0 4 3 | ... | ... | Strychnine 1-20th gr. injected at 3h. 35m. Struggled. |
| Do. | 531 | Do. do. ... | 3 47 0 | 0 1 41 | 0 3 0 | 0 0 32 | 0 6 18 | .. | ... | Strychnine 1-20th gr. injected at 3h. 35m. Struggled. |
| Do. | 532 | Do. do. ... | 4 2 0 | 0 0 59 | 0 1 42 | 0 2 18 | 0 5 31 | ... | ... | Strychnine 1-20th gr. injected at 3h. 43m. Struggled severely. |
| Do. | 533 | Full-grown large-sized pariah ... | 4 15 0 | 0 0 54 | 0 1 43 | 0 1 51 | 0 6 33 | ... | ... | Strychnine 1-10th gr. injected at 4h. 6m. Struggled. |
| Do. | 534 | Full-grown well-nourished pariah. | 4 23 0 | 0 0 48 | 0 1 52 | 0 2 0 | 0 5 30 | ... | ... | Strychnine 1-10th gr. injected at 4h. 12m. Struggled. |
| Dec. 4 | 535 | Full-grown powerful pariah ... | 10 15 0 | 0 1 15 | 0 1 40 | 0 2 0 | 0 4 44 | ... | ... | Strychnine 1-10th gr. injected at 10h. 6m. Struggled very hard. Gasped after the respiration had ceased and before the heart stopped acting. |
| Do. | 536 | Full-grown small pariah ... ... | 10 55 0 | 0 0 58 | 0 1 23 | 0 2 16 | 0 3 2 | ... | ... | Strychnine 3-20th gr. injected at 10h. 45m. Muscles rigid and symptoms of strychnine poisoning commencing when brought on table. |
| Do. | 537 | Full-grown well-nourished pariah. | 11 3 0 | 0 1 18 | 0 2 4 | 0 2 17 | 0 6 0 | ... | ... | Strychnine 3-20th gr. injected at 10h. 55m. Brought on table with symptoms of strychnine poisoning. |

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Do. | 538 | Full-grown small pariah ... ... | 11 12 0 | 0 0 30 | 0 2 0 | 0 2 8 | 0 4 10 | ... | ... | Strychnine 3-20th gr. injected at 11h. 5m. Became convulsed at 11h. 9m. from strychnine poisoning and was chloroformed in this condition. |
| Do. | 539 | Full-grown large-sized pariah ... | 11 16 0 | 0 0 32 | 0 2 58 | 0 3 6 | 0 5 23 | ... | ... | Strychnine 1-5th gr. injected at 11h. 15m. Tetanic convulsions set in in a minute's time, and the dog was at once chloroformed. |
| Do. | 540 | Full-grown pariah ... ... ... | 11 20 0 | 0 0 45 | 0 0 53 | 0 1 56 | 0 5 0 | ... | ... | Strychnine 1-5th injected at 11h. 18m. Tetanic convulsions set in in 1½ minute first time. and the dog was chloroformed in this condition. Gasped 13 times before the heart ceased to beat. |
| Do. | 541 | Full-grown small-sized pariah ... | 11 33 0 | 0 0 42 | 0 2 12 | 0 3 7 | 0 6 53 | ... | ... | Strychnine 1-5th gr. injected at 11h. 23m. Chloroformed during convulsions. |

**III (1)**—*Atropine injected in these cases before the dogs were chloroformed to death with large doses on cloth inhaler.*

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Do. | 542 | Full-grown small-sized pariah ... | 11 41 0 | 0 1 53 | 0 2 10 | 0 3 18 | 0 5 53 | ... | ... | Atropine 1-25th gr. injected at 11h. 30m. Struggled. |
| Do. | 543 | Full-grown well-nourished pariah. | 11 46 0 | 0 0 58 | 0 2 33 | 0 4 0 | 0 6 2 | ... | ... | Do. 1-25th gr. injected at 11h. 35m. Dog struggled. |
| Do. | 544 | Full-grown small pariah ... | 11 52 0 | 0 0 53 | 0 3 0 | 0 3 15 | 0 4 7 | ... | ... | Do. 1-25th gr. injected at 11h. 40m. Dog struggled. |
| Dec. 5 | 545 | Full-grown well-nourished pariah. | 10 13 0 | 0 1 15 | 0 2 45 | 0 3 11 | 0 4 34 | ... | ... | Atropine 1-10th gr. injected at 10h. 3m. Dog struggled during administration. |
| Do. | 546 | Full-grown large but emaciated pariah. | 10 19 0 | 0 43 0 | 0 1 0 | 0 1 28 | 0 3 33 | ... | ... | Atropine 1-10th gr. injected at 10h. 7m. Struggled during the administration. gasped before the heart stopped beating. |
| Do. | 547 | Full-grown fair-sized pariah ... | 10 28 0 | 0 1 21 | 0 3 23 | 0 3 47 | 0 5 59 | ... | ... | Atropine 1-10th gr. injected at 10h. 16m. Dog struggled. |
| Do. | 548 | Full-grown badly-nourished pariah. | 10 37 0 | 0 1 1 | 0 1 46 | 0 2 0 | 0 4 46 | ... | ... | Atropine 1-5th gr. injected at 10h. 23m. Dog struggled. Gasped before death. |
| Do. | 549 | Full-grown emaciated pariah ... | 10 44 0 | 0 1 9 | 0 1 38 | 0 2 2 | 0 4 16 | ... | ... | Atropine 1-5th gr. injected at 10h. 35m. Struggled during the administration. |
| Do. | 550 | Large-sized badly-nourished pariah. | 10 50 0 | 0 0 42 | 0 1 13 | 0 1 20 | 0 5 12 | ... | ... | Atropine 1-5th gr. injected at 10h. 38m. Struggled. Gasped before death occurred. |
| Do. | 551 | Large-sized well-nourished pariah. | 11 0 0 | 0 1 16 | 0 2 18 | 0 2 50 | 0 5 0 | ... | ... | Atropine ⅓ gr. injected at 10h. 48m. Dog struggled. |
| Do. | 552 | Fair-sized well-nourished pariah ... | 11 8 0 | 0 0 48 | 0 1 36 | 0 1 42 | 0 6 18 | ... | ... | Atropine ⅓ gr. injected at 10h. 55m. Struggled very much. Gasped three times before the heart stopped. |
| Do. | 553 | Small-sized very emaciated pariah. | 11 13 0 | 0 1 2 | 0 1 28 | 0 1 32 | 0 2 20 | ... | ... | Atropine ¼ gr. injected at 11h. Struggled in the usual way. As soon as the needle ceased to vibrate, the thorax was opened, and the heart seen to have stopped acting. A post-mortem examination was made, and the heart and other organs found healthy. |

**V**—*Morphine injected in these cases and artificial respiration tried. (The chloroform was administered in the usual way on cloth cap inhaler.)*

| | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|
| Do. | 554 | Large-sized powerful pariah ... | 11 6 0 | 0 1 43 | 0 3 0 | ... | 11 9 30 | 0 8 0 | ... | Morphine ½ gr. injected at 11h. 52m. Dog excited when brought on table and struggled very much during the administration of chloroform. Artificial respiration was commenced 30s. after the breathing had ceased. |
| Do. | 555 | Full-grown well-nourished pariah. | 3 40 0 | 0 0 48 | 0 1 33 | ... | ... | 3 42 3 | 0 5 0 | Morphine ½ gr. injected at 3h. 30m. Artificial respiration was commenced 30s. after the breathing had ceased. Needle in heart at 3m. 47s. ceased to vibrate. Unsuccessful. Struggled during administration. |
| Do. | 556 | Large-sized full-grown pariah ... | 3 51 0 | 0 1 0 | 0 2 11 | ... | ... | 3 53 41 | 0 7 0 | Morphine ½ gr. injected at 3h. 37m. Artificial respiration was commenced 30s. after the breathing had ceased. Dog struggled during the administration. Needle in heart at 4h. ceased to vibrate. Unsuccessful. |
| Do. | 557 | Full-grown fair-sized pariah ... | 4 0 0 | 0 0 53 | 0 1 11 | ... | ... | 4 1 31 | 0 4 0 | Morphine ½ gr. injected at 3m. 50s. Struggled during the administration. Artificial respiration was commenced 20s. after the breathing had ceased. Successful. |
| Do. | 558 | Full-grown well-nourished pariah. | 4 10 0 | 0 0 56 | 0 2 0 | ... | ... | 4 12 20 | 0 5 0 | Morphine ½ gr. injected at 4h. Struggled. Artificial respiration was commenced 20s. after the breathing had ceased. Needle in heart at 4h. 17m. did not vibrate. Unsuccessful. |
| Do. | 559 | Full-grown well-nourished pariah. | 4 21 0 | 0 1 13 | 0 2 17 | ... | ... | 4 23 37 | 0 5 0 | Morphine ½ gr. injected at 4h. 9m. Struggled. Artificial respiration was commenced 20s. after the breathing had ceased. Needle in heart at 4h. 28m. did not vibrate. |

| 1<br>Date. | 2<br>No. | 3<br>Description of dog. | 4<br>Time at which inhalation commenced. | 5<br>Cornea became insensible after | 6<br>Respiration ceased after | 7<br>Pulse stopped after | 8<br>Heart ceased beating after | 9<br>Artificial respiration commenced at | 10<br>Artificial respiration continued for | 11<br>Remarks. |
|---|---|---|---|---|---|---|---|---|---|---|
|  |  |  | h. m. s. | h. m. s. | h. m. s. | h. m. s. | h. m. s. | h. m. s. | h. m. s. |  |
| 1889. |  |  |  |  |  |  |  |  |  |  |
| Dec. 5 | 560 | Full-grown fair-sized pariah ... | 4 32 0 | 0 0 57 | 0 3 14 | ... | ... | 4 35 29 | 0 4 0 | Morphine ½ gr. injected at 4h. 20m. Struggled. Artificial respiration was commenced 15s. after the breathing had ceased. Successful. |
| Do. | 561 | Full-grown emaciated pariah ... | 4 41 0 | 0 1 5 | 0 5 52 | ... | ... | 4 47 17 | 0 3 0 | Morphine ½ gr. injected at 4h. 30m. Struggled. Artificial respiration was commenced 15s. after the breathing had ceased. Successful. |
| Do. | 562 | Full-grown large pariah well nourished. | 4 52 0 | 0 1 3 | 0 1 23 | ... | ... | 4 53 38 | 0 6 0 | Morphine ½ gr. injected at 4h. 40m. Artificial respiration was commenced 15s. after the breathing had ceased. Dog struggled. Unsuccessful. |
| Dec. 6 | 563 | Full-grown well-nourished pariah. | 10 40 0 | 0 0 57 | 0 3 6 | ... | ... | 10 43 36 | 0 10 0 | Morphine ½ gr. injected at 10h. 25m. Struggled. Artificial respiration was commenced 30s. after the breathing had ceased. Unsuccessful. |
| Do. | 564 | Full-grown fair-sized pariah ... | 11 57 0 | 0 1 9 | 0 2 2 | ... | ... | 11 59 32 | 0 6 0 | Morphine ½ gr. injected at 10h. 38m. Struggled. Artificial respiration was commenced 30s. after the breathing had ceased. Successful. This dog died 2m. after it was taken outside and left alone. |
| Do. | 565 | Full-grown well-nourished pariah. | 11 7 0 | 0 1 10 | 0 2 26 | ... | ... | 11 9 56 | 0 4 0 | Morphine ½ gr. injected at 10h. 50m. Struggled. Artificial respiration was commenced 30s. after the respiration had ceased. Needle in heart at 11h. 16m. ceased to vibrate. Unsuccessful. |

**V (b).**—*Artificial respiration tried on a dog poisoned with phosphorus, the chloroform being administered in the usual manner.*

|  |  |  |  |  |  |  |  |  |  |  |
|---|---|---|---|---|---|---|---|---|---|---|
| Dec. 7 | 566 | Full-grown well-nourished pariah, ill from phosphorus poison. | 10 49 0 | 0 0 58 | 0 2 5 | ...... | ...... | 10 51 35 | 0 12 0 | 1 gr. of phosphorus given every morning for 3 days (3rd, 4th, 5th). Did not struggle. Artificial respiration was commenced 30s. after the breathing had ceased. Unsuccessful. |

**I (a).**—*Dogs chloroformed to death with small doses on cloth inhaler.*

|  |  |  |  |  |  |  |  |  |  |  |
|---|---|---|---|---|---|---|---|---|---|---|
| Dec. 9 | 567 | Under-sized ill-nourished pariah ... | 10 32 0 | 0 0 55 | 0 2 55 | 0 3 0 | 0 6 10 | ... | ... | Chloroform administered gradually on a cap with plenty of air. Dog struggled as usual. Gasped 12 times before heart ceased; the last gasp at 10h. 36m. 15s. or 1m. 55s. before heart ceased beating. Weight 20 lbs. |
| Do. | 568 | Full-sized fairly-nourished pariah. | 10 40 20 | 0 1 50 | 0 3 50 | 0 6 40 | 0 8 50 | ... | ... | Chloroformed as in the above case. Dog struggled slightly. Weight 26 lbs. |
| Do. | 569 | Full-sized well-nourished pariah ... | 10 51 0 | 0 1 40 | 0 4 50 | 0 5 2 | 0 9 40 | ... | ... | Chloroformed as in the above case. Dog struggled very much. Weight 26 lbs. |
| Do. | 570 | Do. do. ... | 11 4 30 | 0 0 45 | 0 2 30 | 0 2 35 | 0 6 10 | ... | ... | Chloroformed as in the above case. Dog struggled as usual. Gave one long gasp at 11h. 6m. 45s. or 3m. 55s. before heart ceased. In this case the needle ceased vibrating at 11h. 9m. 15s. when the thorax was opened and the heart found to be contracting until 11h. 10m. 40s. |
| Do. | 571 | Full-grown well-nourished pariah. | 11 14 0 | 0 1 0 | 0 18 31 | 0 19 14 | 0 26 35 | ... | ... | Chloroformed in small doses gradually given with plenty of air in cloth cap. Struggled. Thorax opened in this case and the action of the heart watched to the end. Weight 28 lbs. |

| | | | | | | | | | | | | | | | | | | | | |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Do. | 572 | Full-grown small-sized pariah ... | 11 | 40 | 0 | 0 | 2 | 29 | 0 | 4 | 32 | 0 | 5 | 4 | 0 | 7 | 52 | ... | ... | Chloroform given as in the above case. Struggled feebly. Thorax opened and the heart's action watched to the end. Weight 20 lbs. |
| Do. | 573 | Do. do. ... | 11 | 51 | 0 | 0 | 1 | 33 | 0 | 8 | 13 | 0 | 9 | 0 | 0 | 13 | 28 | ... | ... | Chloroformed as in the previous case. Struggled. Thorax opened after the needle ceased to vibrate and the heart found to have stopped acting. Weight 17 lbs. |
| Do. | 574 | Small and emaciated pariah ... | 12 | 17 | 0 | 0 | 1 | 11 | 0 | 4 | 40 | 0 | 5 | 3 | 0 | 8 | 6 | ... | ... | Chloroformed as in the previous case. Struggled. Thorax opened after the needle ceased vibrating and the heart's action found to have stopped. Weight 18 lbs. |
| Do. | 575 | Small but full-grown pariah ... | 3 | 13 | 0 | 0 | 1 | 13 | 0 | 11 | 3 | 0 | 11 | 55 | 0 | 18 | 22 | ... | ... | Chloroformed in a similar manner. Struggled. Thorax opened after the needle ceased to vibrate. Weight 17½ lbs. |
| Do. | 576 | Full-grown large-sized pariah ... | 3 | 36 | 0 | 0 | 1 | 28 | 0 | 3 | 47 | 0 | 3 | 59 | 0 | 7 | 4 | ... | ... | Chloroformed in a similar manner. Struggled very much. Thorax opened after the needle ceased to vibrate. Weight 28 lbs. |
| Do. | 577 | Full-grown well-nourished pariah.. | 3 | 45 | 0 | 0 | 2 | 16 | 0 | 3 | 0 | 0 | 3 | 15 | 0 | 7 | 11 | ... | ... | Chloroform administered as in the previous case. Struggled. Gasped before the heart ceased to beat. Thorax opened after the needle ceased to vibrate. Weight 21¾ lbs. |
| Do. | 578 | Full-grown large-sized pariah ... | 3 | 56 | 0 | 0 | 1 | 2 | 0 | 10 | 8 | 0 | 10 | 24 | 0 | 14 | 3 | ... | ... | Chloroformed in the same way. Struggled. Weight 30 lbs. |
| Do. | 579 | Full-grown, small and emaciated pariah. | 11 | 6 | 0 | 0 | 3 | 42 | 0 | 8 | 13 | 0 | 8 | 32 | 0 | 12 | 9 | ... | ... | Chloroformed as in No. 578. Struggled. Thorax opened after the needle ceased to vibrate. Weight 20 lbs. |
| Do. | 580 | Full-grown well-nourished pariah. | 11 | 20 | 0 | 0 | 1 | 10 | 0 | 3 | 54 | 0 | 6 | 2 | 0 | 6 | 53 | ... | ... | Chloroformed as in the above case. Struggled severely and had to be held down with great force. Weight 29 lbs. |
| Do. | 581 | Full-grown well-nourished pariah. | 3 | 3 | 0 | 0 | 1 | 52 | 0 | 8 | 4 | 0 | 9 | 0 | 0 | 12 | 8 | ... | ... | Chloroform given as in the above case. Struggled severely and had to be held down with force. |
| Dec. 11 | 582 | Full-grown well-nourished pariah... | 10 | 37 | 0 | 0 | 0 | 58 | 0 | 2 | 15 | 0 | 3 | 3 | 0 | 7 | 14 | ... | ... | Chloroformed in small doses on cloth cap inhaler. Struggled very much, and had to be held down forcibly. Weight 30 lbs. |
| Do. | 583 | Do. do. ... | 10 | 47 | 0 | 0 | 1 | 38 | 0 | 4 | 18 | 0 | 4 | 56 | 0 | 10 | 21 | ... | ... | Chloroformed similarly. Struggled. Weight 26 lbs. |
| Do. | 584 | Full-grown small pariah ... ... | 11 | 1 | 0 | 0 | 1 | 0 | 0 | 4 | 6 | 0 | 4 | 23 | 0 | 13 | 42 | ... | ... | Chloroformed similarly. Struggled. Weight 19 lbs. |
| Do. | 585 | Full-grown large pariah ... ... | 11 | 16 | 0 | 0 | 1 | 7 | 0 | 2 | 5 | 0 | 2 | 47 | 0 | 8 | 50 | ... | ... | Chloroformed similarly. Struggled. Weight 35 lbs. |
| Do. | 586 | Full-grown well-nourished pariah.. | 11 | 27 | 0 | 0 | 1 | 47 | 0 | 4 | 31 | 0 | 4 | 52 | 0 | 8 | 16 | ... | ... | Chloroformed similarly. Struggled very much. Gasped before heart ceased beating. Weight 32 lbs. |
| Do. | 587 | Do. do. ... | 11 | 37 | 0 | 0 | 2 | 51 | 0 | 8 | 41 | 0 | 8 | 59 | 0 | 12 | 4 | ... | ... | Chloroformed similarly. Struggled. Weight 31 lbs. |
| Do. | 588 | Do. do. ... | 11 | 53 | 0 | 0 | 2 | 2 | 0 | 5 | 53 | 0 | 6 | 0 | 0 | 15 | 31 | ... | ... | Chloroformed similarly. Struggled. The respiration returned in this case at 12h. 2m. and continued for 3 m. when the chloroform was renewed in the cap. Weight 28 lbs. |

# PART VII.—METHOD OF EXPERIMENTATION.

## Administration of Anæsthetics.

For experiments on the general action of chloroform the animal was usually placed upon a table and held by several assistants. An inhaler, consisting of a conical bag of cloth containing a sponge or absorbent cotton-wool with the anæsthetic, was then placed over the animal's mouth and nostrils, and kept there as long as necessary. While one observer watched for the loss of reflex from the cornea, the cessation of respiration, stoppage of the pulse, and arrest of the heart, another seated at an adjoining table with a watch before him noted down the times at which each event occurred. The corneal reflex was ascertained by simply touching the cornea with the finger or a blunt instrument, such as the point of an aneurism needle. The cessation of breathing was ascertained by simple ocular inspection of the thorax and diaphragm, the presence or absence of the pulse by feeling the femoral artery with the finger, and the entire stoppage of the heart's action by watching the movements of a needle pushed through the thoracic walls into the heart. The movements of this needle were rendered more evident by a straw bearing a small paper flag being fixed to the end which projected outside the thorax. Animals anæsthetised by this method struggled while they were held until the anæsthetic had had time to take effect. In order to avoid struggling another method was adopted. This consisted in simply lifting the animal into a wooden box 3 ft. 10 in. long by 1 ft. 5 in. broad, and 1 ft. 7 n. deep, and putting on the lid in which was an opening. Through this opening was passed a large piece of blotting-paper on which half an ounce or more of chloroform had been poured. A piece of wood or glass was then placed over the opening in the lid, and in a short time the anæsthetic took effect. To prevent too large admixture of air a strip of spongiopiline was nailed round the edges of the box and covered with vaseline, so that the lid shut down air-tight. Even when a piece of wood was used to close the aperture in the lid at first it was usually replaced by glass, when the chloroform began to take effect, as the movements of the animal could thus be watched. When it fell down insensible it was taken out at once, and if prolonged anæsthesia was required, as for blood-pressure experiments, an additional quantity of the anæsthetic was administered on a cap.

### Administration of Definite Proportions of Chloroform Vapour.

In order to make animals respire air charged with definite proportions of chloroform vapour, the following methods were used ;—

(*a*) A certain quantity, *e.g.*, ½ oz., 1 oz., or 2 oz. of chloroform was poured on blotting paper in the box already described, and when it was seen through the glass in the lid that evaporation was complete, the lid was lifted, the animal introduced, and the lid quickly replaced. The box contained almost exactly eight cubic feet of air, so that the proportion of chloroform vapour in it was easily reckoned. As the whole of the chloroform was evaporated before the animal was introduced, the alteration which its bulk produced in the air-content of the box did not affect the proportion of vapour.

(*b*) But in the process of opening the box and introducing the animal there was almost inevitably some disturbance of the air charged with chloroform which the box contained. In order to avoid this, a box of tin-plate, the same size as the last, was taken, and a round opening 5 in. in diameter was made in one end near the bottom. Round this a collar of tin plate ½ in. in depth was soldered, and on the top of the box was another opening 8 in. long by 5½ in. broad covered by a tin lid. The chloroform was introduced by the opening on the top, and allowed to evaporate as before, and the animal's head was then introduced through the opening at the end, and made as nearly as possible air-tight by a piece of Mackintosh fastened to the tin collar surrounding the opening, and tied round the animal's neck.

(*c*) When it was thought desirable to make the animal inspire a definite proportion of chloroform vapour through a tracheal canula, the same apparatus as above described was used, but in one end of the box two tin tubes were fixed each ¾ in. in diameter. These were connected by India-rubber tubing of a similar bore to the arms of a tin Y tube. The leg of this tube was connected with India-rubber tubing to a glass canula tightly tied into the trachea. By means of India-rubber valves the current of air was made to circulate always in one direction.

(*d*) When it was desired to insufflate the animal's lungs with air containing a definite proportion of chloroform vapour, the apparatus just described was employed with the addition of a bellows, which drew the air from the box. In the valve-hole of the bellows a ¾ in. tube was fixed by means of a large cork bung. This tube was connected by a ¾ in. India-rubber tubing with one of the tubes in the end of the box already described, and by the Y tube with the tracheal canula. A valve over the nozzle of the bellows and another on one arm of the Y tube regulated the direction of the current of air. These valves consisted simply of a piece of dentists' sheet rubber tied loosely over the end of the nozzle or tube and then cut across. When the air passed in one direction, *e.g.*,from the bellows, it blew open the edges of the cut rubber, but when it tried to pass in the opposite direction the edges of the rubber fell together and presented a considerable obstacle.

## Administration of Definite Quantities of Chloroform.

For the purpose of giving a definite quantity of chloroform with unlimited quantity of air another apparatus was used. It consisted of a wide-necked jar 7 in. deep by 5½ in. internal diameter. The top was closed by a tightly-fitting bung, in which there were three apertures. In two of these were fixed ¾ in. glass tubes, and the third was closed by a small stopper, which could be withdrawn at will for the purpose of introducing chloroform into the bottle. To one of these tubes a valve was attached which only allowed the air to enter the bottle, but prevented its exit. The other tube was connected by wide rubber tubing with a tin inhaler. This inhaler was in the form of a truncated cone, and had two ¾ in. tubes opening into it. One of these was at the apical end, and the other at the side of the inhaler. The apical tube was connected with the bottle just described. The side tube was closed by a simple valve consisting of a perforated cork, over which a strip of thin sheet rubber was loosely fastened. At each expiration the rubber allowed the air to escape freely, but at each inspiration it closed completely. The muzzle of the animal was inserted in the inhaler, and a piece of Mackintosh was tied round the inhaler and the head of the animal by means of an elastic bandage, so that the inhaler was nearly air-tight. On inspiration the air passed through the glass cylinder, becoming partially charged with chloroform on its way, and during expiration it passed out through the side tube of the inhaler.

For the purpose of keeping up artificial respiration an ordinary bellows was employed, but to the nozzle of it was fixed a valve which closed when the bellows was pressed, and thus directed the air into the lungs, but opened as the bellows expanded, and thus allowed the air to escape from the lungs.

This valve consisted of a T tin tube ¾ in. in diameter. One end of its transverse limb was connected with the nozzle of the bellows, the other with the tracheal canula. The opening of the perpendicular limb was covered by a piece of tin plate mounted on a hinge in such a way that when the bellows was pressed a cord tied to the upper handle of the bellows pulled it down and closed the opening, but during the expansion of the bellows it was raised and the opening uncovered by the elastic force of a piece of India-rubber. The cord and India-rubber were both attached to a short rod fixed at right angles to that which carried the cover. When it was desired to give chloroform vapour along with the insufflated air, a piece of blotting-paper soaked with chloroform was held over the valve-opening of the bellows, or the opening was closed by a bung and wide rubber tube, and the blotting-paper was placed inside the tube. In the first comparative experiments on the effects of chloroform and ether on the heart in monkeys, when given by insufflation, the bellows and valve just described was employed. Instead of one limb of the T tube being directly connected with the tracheal canula, however, it was connected to a small glass T tube. Each of the other limbs of this tube passed to a long-necked glass flask furnished with a tubulature through which the anæsthetic could be introduced. The neck of the flask was closed by a stopper through which two glass tubes passed, one reaching only a little way below the stopper, but the other extending nearly to the bottom of the flask. One of these tubes was connected with the glass T tube just mentioned, and the other with a glass canula tightly tied into the trachea. At each blast of the bellows the air passed onward through these glass flasks to the trachea of the animals, and returned through the flasks and out through the valve during the expansion of the bellows. It is evident that by this arrangement a certain admixture of ether and chloroform vapour will occur in the tubes between the animals and the valve; so that after a few blasts both animals will receive mixed vapour, although that of chloroform will predominate in the one case and that of ether in the other. In order to avoid this admixture the bellows was used without the valve, and provision was made for allowing the air to escape from the lungs by using a different form of tracheal canula. Instead of a plain glass tube a small glass T tube was used. The perpendicular limb was inserted into the trachea, and one end of the horizontal limb was connected with the bellows; the other was partially closed by a piece of glass tubing, one end of which was drawn out and cut off so as to leave only a small orifice. This was attached by a short piece of India-rubber tubing to the limb of the T tracheal canula. The orifice was of such a size that while it presented sufficient resistance to the passage of air to allow the lungs to become completely inflated when the bellows was pressed, yet during the expansion of the bellows it allowed the air to escape from the lungs and thus prevented over-distension.

When it was desired to give vapour by insufflation to one small animal, such as a monkey or rabbit, only the India-rubber ball of Junker's apparatus was used instead of the bellows. Junker's apparatus consists of the India-rubber balls ordinarily used for spray producers, but these are attached to a bottle in which the anæsthetic is put in such a way that, instead of producing spray, the air simply passes through the anæsthetic and becomes charged with the vapour. It then passes to a mouth-piece and is inhaled. The mouth-piece which we employed for dogs was made of stout leather in the form of a truncated cone. A metal tube ¼ in. in diameter, opening into the inhaler at its apical end, conveyed the vapour into it. This tube fitted the opening in the inhaler, through which it passed closely, but the junction was not air-tight. The inhaler at its other end did not fit the dog's muzzle at all closely, so that there was a free circulation of air. When we wished to prevent this and to give concentrated vapour, a piece of Mackintosh was tied over the inhaler and muzzle so as to close up the space between them.

### Blood-pressure Experiments.

In all these experiments the pressure within the arteries was registered by one of Ludwig's kymographs consisting of a mercurial manometer and a revolving cylinder on which the oscillations of the mercury were recorded. The paper on the cylinder was smoked by means of burning camphor. The float on the mercury of the manometer consisted of a cylindrical piece of ivory tapering below and bearing above a steel wire, near the top of which a writing point of glass was fixed by a bit of cork. This point was kept in contact with the smoked paper by a silk thread loaded with a shot. To the top of the manometer was fastened a second writing point made of glass or of copper foil. When the mercury was at zero this point was at the same level as the point on the float, and as the cylinder revolved it traced the zero line round it and thus afforded a means of estimating the height of the blood-pressure at any moment. The cylinder revolved once in nearly thirty minutes. Near the top of the cylinder was placed a magnetic time-marker worked by a DuBois Reymond's key.

An observer sat constantly beside the cylinder with a watch in front of him, and recorded on the cylinder by means of the time-marker, as well as the instant when anything worthy of note occurred, the time, minute by minute. When the revolution of the cylinder or drum was complete it was removed, and a second drum, which was kept ready smoked, was substituted. The tracing on the first drum was at once cut off and varnished, and another paper put on and smoked, so as to be in readiness when required.

The movement of the cylinder in these experiments is much slower than that usually employed, but it has the double advantage of allowing the whole record of the experiment to be reproduced and of rendering distinct even small variations of pressure which are apt to escape observation in the long tracing taken on a rapidly revolving drum. But the tracing taken on such a slow drum as we employed has the disadvantage of being so compressed that the individual pluse-beats are invisible.

To obviate this defect a method of double registration was adopted, which had been devised by one of us for a previous research. In the connection between Ludwig's kymograph and the artery a Y tube was inserted, by means of which a second kymograph, the cylinder of which revolved once in 3m. 9s., or about ten times as rapidly as the first could also be brought into communication with the artery. Both were provided with stop-cocks, so that they might either be allowed to work simultaneously, or the communication of either or both with the artery cut off at will. If a mercurial manometer had been used in the second kymograph it would have given the number of pulse-beats, but the oscillation of the mercury itself would have modified their size and form. One of Fick's spring manometers was therefore used, and in order to prevent the oscillations of the mercury in the other manometer affecting Fick's the stop-cock connecting the former with the artery was almost always turned off when the latter was turned on. This plan had the further advantage that the straight line traced by the float of Ludwig's kymograph attracts the eye so that a glance at the tracing at once shows when a tracing has been taken by Fick's manometer, and by attaching corresponding numbers to these blanks in the tracing from Ludwig's kymograph and the tracings by Fick's kymograph, the relation of the tracings to each other can be at once ascertained. The clock-work of Fick's kymograph was usually started at the moment when the stop-cock connecting it with the artery was opened. A short time is required for the clock-work to attain its full speed, and therefore at the beginning of a tracing the oscillations may be closer together, and the pulse may appear to be quicker at the beginning than the end of a reading of Fick's kymograph, although the pulse rate might be really the same throughout. The cylinder was usually, though not always, allowed to revolve a little space after the connection of the manometer with the artery had been cut off in order that the readings might be more distinctly separated from one another. The mercurial manometer was connected with the artery partly by metal and partly by India-rubber tubing. The Fick's manometer was connected entirely by India-rubber tubing in the experiments up to November 5th, but after that the connection was made partly by India-rubber and partly by metal, exactly as in the Ludwig's. The whole of the connecting tubes, except those between the Y tube and the manometers where water was used, were filled with a solution of sodium bicarbonate in order to prevent coagulation occurring in them.

This solution was made by saturating boiling water with sodium bicarbonate and then boiling it for some minutes, so as partly to convert it into carbonate. It was then allowed to cool, and was poured into a large glass vessel furnished with a tubulature near the bottom, and suspended on the wall of the room at a height of 7 ft. from the floor and 4½ ft. above the level of the table. From the tubulature of this vessel the solution was conveyed by India-rubber tubing to a glass T-piece inserted in the connection between the manometers and the artery. By means of clamps the soda solution could be passed at will through the tubes leading to the artery. Before beginning an experiment the pressure within the manometer was raised by means of the soda solution to a height approximating to the estimated blood-pressure in the animal so as to prevent the blood from passing too far up the tubes and forming clots. This was also done whenever the artery and manometer had to be disconnected on account of clots having formed. In order further to lessen the tendency to form clots an elongated glass bulb was introduced into the tubing, and was connected by a short piece of India-rubber tube with the glass canula in the artery. The canulas used for insertion into the artery were blown with a short neck to hold the ligature and prevent the canula from slipping out of the artery. They were of various sizes so as to fit the artery of the animal experimented on. In all the experiments, with one exception, the animal was first rendered insensible by being placed in a box with chloroform, as ether given in this way would have been useless. As soon as it became insensible it was taken down and the legs fastened with cords to a simple piece of board 4 ft. 6 in. long by 2 ft. 2 in. broad. To one end of this one of Bernard's dog-holders was attached and in this the animal's head was put so as to hold it still. Chloroform or ether was then administered on a nose-cap as required to keep the animal anæsthetised and one carotid exposed, the left being selected in every case as it was nearest to the manometer. Occasionally one or both vagi were also exposed and a loop of thread placed under them, but not tightened round them.

The artery was firmly ligatured on its distal side, and the proximal side was compressed by spring forceps covered with India-rubber tubing to prevent their injuring the coats of the artery. The artery was then opened, the canula inserted, and firmly tied in. The canula was then filled with soda solution by means of a pipette, and connected with the glass bulb already mentioned, care being taken that no air bubbles were present. The spring forceps were then removed from the artery, the stop-cock of the manometer turned so as to open the connection with the artery, and the tracing commenced. All this time the animal was kept unconscious by the administration of an anæsthetic from time to time as required.

### Registration of the Movements of the Heart.

These were sometimes registered by inserting a needle into the heart, and connecting a thread to the end which projected outside the thorax when this was unopened, or outside the cardiac walls when the thorax was opened and the heart exposed. This thread was in the first instance tied to the lever of a Marey's tambour (*A*), and the movement transmitted by it to a second one (*B*), which recorded it. The resistance of the two tambours, however, was too great, and the tracings obtained were unsatisfactory. Better results were obtained by connecting the needle in the heart with a time-marker by means of two very light wooden pulleys. The lever of the time-markers being kept up by a very fine spring, a very slight pull sufficed to depress it, and thus it marked even when the cardiac action was weak, and had the further advantage of opposing very little resistance to the action of the heart.

### Registration of the Respiration.

For this purpose the two Marey's tambours *A* and *B* were used. *A* was usually connected to a needle pushed through the chest walls into the diaphragm. On one or two occasions an incision was made just under the margin of the ribs, and a piece of thick copper-wire flattened at one end, was introduced, so that the flat pieces lay between the diaphragm and liver. The other end projected outside, and the thread from Marey's tambour was fastened to it. In some of the later experiments a fish-hook was used instead of a needle.

# PART VIII.—THE CONTROVERSY ON ANÆSTHETICS AND THE HYDERABAD COMMISSION.

In this part of the report it is deemed advisable to reproduce, prefaced by Syme's lecture, every thing of importance bearing on anæsthetics and the Hyderabad Commission, which has been published between the time when the conclusions of the Commission were put forward in *The Lancet*, and the present publication of their tracings and experiments. The Commission has always courted criticism, and has frankly recognized that the chloroform question can only be settled by full and free discussion; and the soundness of this conviction is illustrated by the primary disagreement between the Hyderabad Commission and the Glasgow Committee. The Glasgow Committee inferred from their research that the chief dangers of chloroform are reduction of blood-pressure and sudden stoppage of the heart. Their animals, however, did not die, and the ground for this inference, which was drawn from a variety of falls of the blood-pressure in their experiments, is the universal belief that a general fall of blood-pressure is in itself a source of danger. The Hyderabad Commission has proved that stoppage of the heart under chloroform may be a safeguard against poisoning or overdosing; and this discovery is irreconcilable with the finding of the Glasgow Committee. But prolonged consideration and ventilation of the matter have made it clear that a general fall of the blood-pressure is not necessarily a sign of weakening of the heart; and it is therefore at once evident that the Glasgow and Hyderabad experiments are in complete accord. Both demonstrate that sudden and gradual falls of blood-pressure may occur under chloroform without danger to the organism; and the fact that a general fall of the blood-pressure, whether gradual or sudden, is not in itself dangerous, forms in reality the physiological foundation of the clinical truth that normal chloroform anæsthesia is free from risk. Nothing but good can arise from the controversy; and accordingly the agreement now established, between the experimental data of the Glasgow Committee and those of the Hyderabad Commission, has only to be extended to their conclusions to be the means of settling the chloroform question and saving thousands of lives.

# LECTURE ON CLINICAL SURGERY DELIVERED DURING THE WINTER SESSION OF 1854-55 BY JAMES SYME, Esq., PROFESSOR OF CLINICAL SURGERY IN THE UNIVERSITY OF EDINBURGH.

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*From "The Lancet," dated 20th January 1885.*

## CHLOROFORM.

I HAVE now to speak of some cases in which chloroform will be given as usual, in consequence of the pain otherwise attendant on the operations to be performed; but before the patients are brought in I may take this opportunity of saying a few words regarding the use of chloroform, as you see that fatal cases, I am sorry to say, still occur, and that the medical journals, consequently, express doubts as to the use of chloroform at all, or say that, if used, it must be only with the greatest caution. Chloroform is no doubt a very powerful agent, sufficient to destroy the strongest individual if employed freely enough. Its fatal effects were shown at an early period in the following way: a lecturer in London was illustrating its action upon a guineapig, which was placed under a glass jar along with some of the anæsthetic: the professor, in the eagerness of his discourse, left the animal too long under the jar, and it died. He explained to his audience the cause of the accident, but, though the reasons he gave were satisfactory, yet an impression was made upon the public which was not soon effaced. In this respect, however, there is nothing peculiar to chloroform as a medicinal agent; opium, prussic acid, strychnia, &c., not only may, but often do, destroy life, through being used in over-doses. But the question is, may it be used judiciously so as to do the good without exposing the patient to the risk of the evil? It is said in London that it cannot; that the risk is so great that it is only justifiable to use it in case of operations accompanied with an extreme degree of pain, or where stillness on the part of the patient is essential to success, and that the greatest caution is required in its administration; here we say that, if used with moderate care, it is perfectly safe. It was in this theatre that chloroform was first administered in public, by Dr. Simpson, seven years ago; since then it has been almost daily given here, yet we have not had a fatal case. It is true one solitary instance of death from chloroform has occurred in another part of the hospital, but that case has nothing to do with us ; so far as my department is concerned, it might as well have been at Guy's Hospital or in Kamschatka, or anywhere else ; indeed, it so happened that at the very time when that unfortunate event was taking place in another part of the establishment, I was myself performing an operation on a patient under chloroform in this theatre.

In inquiring into the reason of this difference between the experience of chloroform in London and here, we have not far to search for the explanation ; it must lie in one of three things, *viz.*, difference in the chloroform, difference in the patients, or difference in the mode of administration : with regard to the chloroform, I believe, that most of that which is used in London is made in Edinburgh, and I know that some of the fatal cases might be shown to have occurred with Edinburgh chloroform.

With respect to the patients, it appears that great care is taken in London to use chloroform only in persons free from chest affections, especially cardiac derangements : here we never ask any questions as to the state of the heart or constitution of the patients. In all cases where chloroform is required for an operation, it is freely given. Now, considering the frequency of cardiac disease, and particularly of fatty heart,—which, in fact, is I believe rarely absent, at any rate in elderly persons,—and considering also the immense number of patients operated on, you cannot doubt that many hundreds with fatty degeneration of the heart have had chloroform administered to them here ; we even give chloroform without scruple, where we know disease of the heart exists. Within the last week, a case in point occurred in my practice. A patient, with great dread of pain, and also with a horror of chloroform, had long endured severe pain from his disease, till existence had become a burden, because he could not venture to undergo the necessary operation without chloroform, while his medical adviser considered that to take it would be for him almost certain death, on account of organic disease known to exist in the heart, for which he had consulted, and which it may be remarked had been recognized by Dr. Addison of Guy's Hospital. At length his medical attendant said to him, that he had suffered so much from his complaint, that even if he died under chloroform this would be better than remaining as he was ; while, if it should so happen that he should recover, he would be able to enjoy the rest of his life free from the disease. The patient could not resist the force of this argument, and came to Edinburgh prepared for either alternative. I performed the operation under chloroform ; and the first thing that he did on waking was to ask for a cigar.

As another example, I may mention the case of an old gentleman, aged seventy-four, affected with disease of the heart, from whose bladder I removed a large stone, some years ago. Chloroform was administered as he lay in bed; and he was put so fully under its influence, that he was taken from the bed, was operated on, and put back to bed, before he woke from his sleep. He recovered perfectly; but some time afterwards died and Dr. Begbie, on examining the body, found the disease of the heart, which had been diagnosed during life. We cannot therefore attribute the absence of deaths here to our being more discriminating than others in the patients to whom we administer chloroform; the very reverse, in fact, being the case.

I think then, gentlemen, that we are necessarily led to the conclusion that the difference of results depends on difference in the mode of administration. We know that in other cases differences in the method of procedure had led to differences of results, *e.g.*, it was said in London that amputation at the ankle invariably caused sloughing; but it turned out that the surgeon who made this statement performed the operation in a manner that deviated much from the principles on which it is here performed successfully, and which led inevitably to sloughing. So of dividing stricture by external incision, it was said that the extravasation of urine must necessarily take place in a dangerous manner, and that other serious complications may occur; but it turned out that the incision had been made without guide on a silver catheter; in short, with such deviations from principles as fully to account for the results.

So far as I can ascertain, from what I have heard and read upon the subject, there are important differences between the mode of administration of chloroform here and in London. It appears that here it is given according to principle, there according to rule. There great attention is paid to the number of drachms or minims employed; here we are entirely regardless of the amount used, and are guided only by the symptoms of the patient. The points that we consider of the greatest importance in the administration of chloroform are, first, a free admixture of air with the vapour of the chloroform, to ensure which a soft porous material, such as a folded towel or handkerchief, is employed, presenting a pretty large surface, instead of a small piece of lint or any other apparatus held to the nose; secondly, if this is attended to the more rapidly the chloroform is given, the better, till the effect is produced; and hence, we do not stint the quantity of chloroform. Then—and this is a most important point—we are guided as to the effect, not by the circulation but entirely by the respiration ; you never see anybody here with his finger on the pulse while chloroform is given. So soon as the breathing becomes stertorous, we cease the administration ; from what I have learned it is sometimes pushed further elsewhere, but this we consider in the highest degree dangerous. Attention to the tongue is another point which we find of great consequence. When respiration becomes difficult, or ceases, we open the mouth, seize the tip of the tongue with artery-forceps, and pull it well forward ; and there can be little doubt that death would have occurred in some cases if it had not been for the use of this expedient. We also always give the chloroform in the horizontal position, and take care that there is no article of clothing constricting the neck. There are thus considerable differences between our practice and that which prevails more or less elsewhere. We use no apparatus whatever, take the respiration for our guide, attend to the condition of the tongue, and never continue beyond the point when the patient is fully under the influence of the anæsthetic.

You observe that in this matter I am very far from taking any credit to myself ; all that I have done has been to follow the example of Dr. Simpson, and all that I would say respecting our brethren in London, is, that they have not been so fortunate as to get into the right way in the first instance, and I would urge upon them to banish all previous notions, and to keep in view the essential points to which I have alluded ; then, if unfortunately there should still be fatal cases, I shall not presume to speak further upon the subject. As the matter at present stands, the discussions prevalent in the profession tend to give the public a dread of chloroform, and to limit the advantages which it confers ; and so long as the difference of opinion seemed due to important difference of practice, I felt called upon to address to you the observations I have made.

# THE CHLOROFORM COMMISSION IN INDIA.

[*Reprint of an article from the "Pioneer" of November 6th,* 1889.]

The arrival of Dr. Lauder Brunton and Surgeon-Major Bomford in Hyderabad, to repeat and extend the chloroform experiments which were carried out last year in the Nizam's capital, is of interest not only to the medical profession, but to the public generally : and some account of the scope of their investigations and their bearing on the practice of surgery may not be out of place. Chloroform was introduced to the medical profession by the late Sir J. Y. Simpson of Edinburgh, who first employed it in midwifery in the year 1847. It was very soon afterwards used in surgery in Edinburgh by the late Mr. Syme, and its position as an anæsthetic quickly became established. It was some little time before Syme could lay down precisely the principles on which the safe administration of chloroform depends, and meanwhile deaths occurred elsewhere, discussion arose in the profession as to their cause, and the public became alarmed and acquired a dread of chloroform which thenceforth limited its advantages.

In the year 1855 Mr. Syme published a lecture in the *Lancet* in which he showed that there was an important difference in practice in the administration of chloroform in Edinburgh and in London. In Edinburgh, surgeons were guided as to the effect of chloroform entirely by the respiration ; in London, partly by the respiration and partly by the circulation, so that it was, and still is, the rule in London to watch the pulse carefully throughout the administration. The teaching by the two schools amounted to this : in Edinburgh it is held that chloroform never affects the heart directly, and that signs are always given by the respiration whereby danger can invariably be averted ; in London it was taught that one, if not the greatest, danger of chloroform lies in the fact that it sometimes affects the heart directly, and that its administration is never free from risk. Syme's lecture was couched in polemic terms and increased the divergence of opinion between the Edinburgh and the London surgeons, and from time to time deaths from chloroform continued to occur. In 1864 a Commission was appointed by the Royal Medical and Chirurgical Society to enquire into the action of chloroform with special reference to its effect upon the heart. This Commission performed few reliable experiments, but nevertheless arrived at the conclusion that chloroform does directly affect the heart. In 1870 Syme and Simpson died, still adhering to their original opinion and never having had a fatal accident with chloroform. Between 1870 and 1880 Syme's principles were gradually lost sight of, and the opinion that chloroform affects the heart gained ground, and was fortified by another Commission which was appointed in Glasgow in 1879, and, like the previous one, did not hesitate to condemn chloroform upon altogether insufficient data. As the belief in the safety of chloroform has decreased, two remarkable phenomena have come about : (1) deaths from chloroform have increased in exact proportion to the amount of attention bestowed upon the heart and circulation ; and (2) an effort has been made to substitute there or some other anæsthetic for chloroform.

So far no anæsthetic has been discovered which can be favourably compared in any way with chloroform administered on Syme's principles. Ether can be used only in certain climates and under certain conditions. Nitrous oxide is said to induce epilepsy and insanity, and none of the other so-called anæsthetics deserves any consideration at all. A further evil result of the morbid and unfounded dread of chloroform fostered by the English and Scotch Commissions has shown itself in many of the large medical schools and hospitals in Great Britain, where anæsthesia has been relegated to specialists, and students have ceased to enjoy the advantages of practical teaching in anæsthetics, so that they now begin practice almost ignorant of a subject in which they ought to have become experts before they left college. Syme's pupils, however, have not abandoned his precepts, and it is a notorious fact that they have had the same success with chloroform as he had. For example, in both the hospitals attached to the Hyderabad Medical School, chloroform is always given by students with absolute safety, and with no precautions whatever against failure of the heart except attention to the respiration. There can be no doubt that immunity from danger with chloroform in Hyderabad is not due to any peculiarity of climate, or of the patients, or to the quality of the drug, but simply to the manner in which it is administered.

In 1888 the Government of the Nizam of Hyderabad appointed a commission to enquire into the alleged dangers of chloroform; and the report drawn up by this body, based on numerous experiments, tended to prove that chloroform does not affect the heart. The Minister of Hyderabad, Sir Asman Jah, thereupon offered the *Lancet*, £1,000 to send a representative to India to repeat and extend the experiments of the first Hyderabad Commission. This offer was accepted, and Dr. Lauder Brunton, F.R.S., has arrived in Hyderabad, and with Surgeon-Major Bomford, M.D., London, of the Indian Medical Service, has constituted a second Commission, which it is hoped will settle finally whether chloroform can be given so as to do good without the risk of evil. Nothing can show better the beneficent nature of the solid and friendly tie which binds together the British and the Nizam's Governments than the remarkable and cosmopolitan interest which H. H. the Nizam and his officials, one and all, have taken in bringing the question of the safety of chloroform to the proof. It redounds to the honour of the Government of India, no less than to that of the administration of the Prime Minister, Sir Asman Jah, that the leading Indian State should take a principal share in the settlement of a question which concerns the welfare and the happiness of the whole mankind.

## LEADING ARTICLE.

"*The Lancet*," *January* 18*th*, 1890.

The second Hyderabad Chloroform Commission, whose report appears on another page of "*The Lancet*" of to-day's issue, has excited a great deal of interest, both on account of the importance of the subject of investigation and the circumstances under which the work has been undertaken. The Commission owed its existence to Surgeon-Major Lawrie's veneration for his late teacher, Professor Syme, and his desire to prove the correctness of Syme's teaching that chloroform might be with perfect safety administered, provided the administrator watched the respiration with sufficient care. Surgeon-Major Lawrie's own experience of many thousand cases confirmed, in his opinion, the truth of this doctrine, but it had been impugned on account of the results of physiological experiments, and he was therefore desirous of proving its truth by laboratory experiments as well as by clinical experience. Accordingly, at his suggestion, the Nizam's Government appointed a Commission consisting of Dr. Hehir and Messrs. Kelly and Chamarette. These gentlemen, from a series of experiments, arrived at the conclusion that chloroform always arrests the respiration before the heart. A copy of their report and of some remarks upon it by Dr. Lawrie were forwarded to "*The Lancet*," but the facts brought forward seemed to us insufficient to overthrow the conclusions of many observers both in this country and abroad whose researches seemed to prove that one of the dangers from chloroform was paralysis of the heart. Our hesitation to accept the conclusions of the first Hyderabad Commission led Dr. Lawrie to propose a second one, to which "*The Lancet*" should send a representative, and for this purpose the Nizam's Government most generously offered to contribute the sum of £1,000. We accordingly selected Dr. Lauder Brunton, F.R.S., and on his arrival at Hyderabad on October 22nd, a second Commission was at once formed, consisting of Surgeon-Major Lawrie as President and of Drs. Lauder Brunton, Bomford, and Rustomji as members. The members of the first Commission were also associated in the work and rendered most valuable assistance. The experiments were of two kinds; those of one group being made without recording apparatus, and being intended to ascertain what influence is exerted by various conditions upon the relation between the stoppage of heart and of respiration, and the limits within which artificial respiration and other means of resuscitation are useful. The second group consisted of experiments with recording apparatus, and were made for the purpose of ascertaining the effect of various conditions upon the heart and blood-pressure. In the first group chloroform was given in all sorts of ways, alone or with morphine, atropine, and strychnine, to animals healthy and diseased, fasting or replete. The result was invariable: in every case the respiration stopped before the heart, sometimes a long time before it. But the effect of partial asphyxia in causing the heart to stop very soon after the respiration is deserving of particular notice.

The second group of experiments on heart and blood-pressure was made with apparatus arranged in such a way that the whole experiment could be recorded from beginning to end in such a compass as to admit of photographic reproduction in its entirety. This was managed by recording the general blood-pressure on a slowly revolving drum, and taking at intervals a tracing on a second drum, revolving with sufficient rapidity to show each beat of the pulse. About one hundred and fifty experiments were made in this way, and the influence of everything that seemed likely to affect the blood-pressure during chloroform narcosis was ascertained. Particular attention was directed to the production of shock or syncope, and to the effect of chloroform itself on the heart and blood-pressure in healthy animals, and also in cases where fatty degeneration of the heart and other organs had been produced by the previous administration of phosphorus. The results of these experiments were unexpected. It was found to be exceedingly difficult to affect the heart reflexly, and recourse was therefore had to direct stimulation of the vagus, by which the heart could be slowed or stopped completely. Instead of this causing the death of the animal, however, it appeared rather to be a safeguard, preventing the anæsthetic from being conveyed in too great quantities to the nerve centres.

In our note to one of Dr. Brunton's telegrams we remarked on the change which his views had undergone. From the report we now publish it will be seen that the discrepancy between the views of different schools arises from the fact that sufficient consideration has not been given to the conditions under which the chloroform is given. Although it may paralyse the heart if applied directly to it, yet this condition does not occur in practice, for here it is neither applied to that organ nor yet is it blown forcibly into the lungs. It is inhaled by the patient, and when this is the case it stops the respiration before the heart. The practical outcome of the research would appear to be that deaths from chloroform are not inevitable. They are therefore preventable, and by due care in its administration they may be with certainty avoided. The conclusions of the Commission are sweeping, and without abundant evidence cannot be accepted. The most important tracings on which they are founded will be published in "*The Lancet*," but it would be impossible to give them all, amounting as they do to about six hundred. Fortunately the generosity of the Nizam's Government has not been limited to the appointment and payment of the Commission, but by well-timed liberality it has secured the permanent utility of the work done by it, for it has had every tracing photographically reproduced, and will present a copy of the complete work to all the principal medical libraries throughout the world. For this generous liberality the thanks of all mankind are due to the Nizam and to his able and enlightened Minister, Sir Asman Jah, as well as to the other members of his Government who have by their co-operation aided and facilitated the work.

# THE HYDERABAD CHLOROFORM COMMISSION.

## LETTER FROM SURGEON-MAJOR A. DEANE.

To the Editors of "The Lancet."

SIRS,—Having followed the proceedings of the Hyderabad Chloroform Commission with much interest from the beginning, I trust you will allow me space to draw attention to what, if its dicta be accepted in their entirety, must be an important outcome of its conclusions.

The final conclusion of the Commission is that chloroform may be administered *in any case** with ease and *absolute safety* (the italics are mine) if its directions for administration be adhered to. With this conclusion I entirely disagree, as I know from my own personal experience that people do exist who cannot tolerate chloroform as an anæsthetic, no matter how it is administered, and yet, unfortunately, we cannot detect anything abnormal about them until it is being given when either death or a condition very analogous to it ensues. I will relate the history of two such cases later on. Now, suppose a death were to occur from chloroform after the pro-

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[ * The conclusion of the Commission is that chloroform may be administered in any case *which is fit for an operation.*]

mulgation of this report, what would be the result to the administrator? Would a coroner's jury find death from misadventure or would the verdict be one of manslaughter? I know that I should be very sorry to administer chloroform if I knew I was liable to be found guilty of manslaughter should the patient die under it, and I fancy that many other doctors would say the same. For the last fourteen years I have administered chloroform precisely as the Commission recommends, and although I never lost a patient under it, still I went so near doing so twice that my experience may be interesting. About twelve years ago, when civil surgeon of a very large district in India, I had a great deal of surgery, and so great was the crush of people coming daily that at last it became almost impossible to get through the work. To enable me to cope with it I used to make my assistant surgeon give chloroform to one patient while I went on examining others in order to pick out those cases that were suitable for operation. I worked in a verandah only a few yards from the door of the operating-room, and one day my assistant surgeon rushed out to tell me that the patient on the table to whom he was administering chloroform was dead. In a moment I reached the room, and at once commenced trying to resuscitate the patient, and I succeeded after about twenty minutes of the hardest work I ever had. My assistant surgeon assured me that he had given very little chloroform, when suddenly the man stopped breathing and at once passed into the alarming condition in which I found him. He (my assistant) was a very capable man, of great experience in giving chloroform, so that I had no reason to doubt his word. Moreover, he had helped me to give chloroform hundreds of times, and knew how to give it just as well as I did. Some two years afterwards I was giving chloroform to an old woman, and she had only taken a few whiffs, not enough to harm an infant of a week old, when suddenly, without the least previous warning, she ceased breathing and became to all appearances dead, and it took me all I knew to get her round again. There was nothing whatever in her previous condition to lead one to suppose that she could not tolerate chloroform, and I know that no one could have given it more carefully than I did. I say, therefore, that one certainly cannot give chloroform with absolute safety in every case.

I do not write this to find fault with the general findings of the Commission. On the contrary, I know from my own experience that they are absolutely true, and I rejoice that at last the truth is known about chloroform, and that it is to a man of my own service the credit is due for this crowning victory in its favour. I only say that I am not satisfied as to its absolute safety in every case, and I shall be glad to hear whether my experience of it can be borne out by others.

I am, Sirs, yours faithfully,
A. DEANE, M.D.,
Surgeon-Major, Bengal Med. Service.

CROYDON, Jan. 20th, 1890.

# LETTER FROM DR. E. F. NEVE.

## TO THE EDITORS OF "THE LANCET."

SIRS,—The results of the work of the Hyderabad Commissions, important as far as they go, can only be considered as final on one division of the subject. Where the nervous system is an important factor, the difference between the highly-organised cerebro-spinal system of man as compared with animals is not likely to be overlooked. Sometimes the results of clinical experience may disagree with those of physiological experiment. In such cases practical men are apt to prefer the former. But even in clinical work sources of error exist. There are three such which ought to be kept in view:—(1) That those in many respects most qualified to judge in the question, *viz.*, experts, anæsthetists by profession, are from their very position apt to have a distinct bias; for of the essence of the controversy is the contention on the one hand, that average ability, great care, and moderate experience are sufficient, for the safe administration of chloroform, without any special apparatus. Opposed to this is the reiterated belief, supported of course by the united body of professional anæsthetists, that both experts and apparatus are required, whether for chloroform or ether. (2) In favour of the use of chloroform as a routine anæsthetic is the weight of conservatism, including the convenience of administration of the drug. (3) In cases of death under administration, there is a natural tendency for even an upright and honest man to seek for causes of death outside the obvious possibility of accidental asphyxia from over-dosage or failure in resuscitation. A question in which such conflicting interests are involved can only be settled by facts. The Hyderabad Commission has contributed its quota. When chloroform was first introduced its use was attended for a long time by a wonderful freedom from mishaps. Of late years deaths have increased, and *pari passu* a certain degree of alarm has arisen? But are the deaths the cause of the alarm or is the alarm the cause of some of the deaths. The latter view is by no means untenable. People may die of pure fright or of ordinary syncope. The state of anæsthesia may increase the possibility of such an untoward occurrence, which may take place under even nitrous oxide.* If this be so, then a certain responsibility rests on all who foster such an alarm by unguardedly condemning the use of a popular anæsthetic. But, after all, the crucial question is—What is the mortality in the practice of successful operators? This is the test which we apply to all important surgical procedure. By it we eliminate many fallacies.

Now, with regard to the practice of the Kashmir Mission Hospital, I may state that there has never been a death under chloroform. Since 1877, the number of surgical operations performed has been 13,275. In connection with these, at the lowest estimate, chloroform has been administered more than 5,000 times. Many surgeons in Great Britain must have had vastly greater experience. If it be possible to accumulate facts from such sources, we shall soon be in a position to judge as to the intrinsic mortality of this, in one sense, the most important operation in surgery.

I am, Sirs, your faithfully,
ERNEST F. NEVE, M.D., F.R.C.S. ED.

KASHMIR, December 13th, 1889.

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* *The Lancet*, vol. II., 1889, page 804.

## ANNOTATION FROM *THE LANCET*, JANUARY 25TH, 1890.

In our annotation on the report of the first Hyderabad Chloroform Commission in our issue of March 2nd, 1889, we strongly deprecated anything like slipshod carelessness in the administration of chloroform, or anything likely to lead to such carelessness. Nothing, we are certain, could have been farther from the minds of either of the Commissions than to inculcate carelessness, and a careful perusal of the practical conclusions which we printed last week will show how necessary the Commissions consider care to be, and how stringently they inculcate it. Yet one of our contemporaries, in referring to the report of the Commission, says: " Henceforward chloroform is as safe as a glass of whisky-and-water. That is the net result of the Hyderabad Chloroform Commission." This may be true of chloroform carefully administered, but the least carelessness or inattention may lead to the most serious results, as was well shown by the cases of accidental death from chloroform amongst the animals used by the Commission. We think the instance is worthy of particular note in which the chloroformist was present in body but absent in mind, and allowed his attention to wander from the respiration of the animal to the performance of the operation. Instead of being an immense relief to every practitioner who has to administer chloroform, the conclusions of the Hyderabad Commission will have precisely the opposite effect. For if a death occurs during the administration of chloroform at present, the administrator may satisfy himself that such death was due to the effect of chloroform on the heart, and was unavoidable. But if the conclusions of the Hyderabad Commission are correct, this will no longer be possible, for their deliberate conclusion is that deaths from chloroform are preventable, and that their occurrence in the future will indicate carelessness in the chloroformist. It would be monstrous to make such a conclusion retrospective, and to say that all deaths that have already occurred were due to carelessness, because imperfect knowledge leads to a similar result, and it is only when the exact dangers and mode of obviating them become known that the responsibility of avoiding them arises. No one thinks of blaming a commander when his ship strikes a sunken rock not marked on his chart, but when once the danger has been clearly indicated he is regarded as blameworthy if he does not avoid it. The work of the Chloroform Commission has not altered the properties of chloroform in the least; an over-dose will kill as readily as ever, but we trust their attempts to define more clearly the way in which it kills, and the mode in which death may be avoided, will have the desired effect of preventing deaths in the future, just as marking the situation of a sunken rock will prevent further shipwrecks.—*The Lancet*, 25th January 1890.

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## LETTER FROM MR. WOODHOUSE BRAINE.

### TO THE EDITORS OF "THE LANCET."

SIRS,—It may appear almost ungenerous to criticise the conclusions at which the Hyderabad Commission has arrived, when all its members have thrown themselves heart and soul into the labour, and have spared no pains to get at the truth of the great question as to whether chloroform ever produces a fatal result by cardiac failure before the respiratory functions are affected; but if I remain silent I shall probably be considered as acquiescing in those conclusions, and therefore I trouble you with this letter.

I greatly fear that, as a result of the Hyderabad Commission, the opinion that death from chloroform is always prefaced by some change or sign of danger in the patient's breathing will gain ground. But does the Commission prove this? I think not. What it does prove is that in dogs and monkeys and some other animals, in India, respiration always gives warning of an overdose and ceases from one to ten minutes before the heart stops for ever. This is no new statement, for in Snow's work on Anæsthetics, published in 1857, we find the following passage: "The greater number of experimenters who have killed animals with chloroform have found that the action of the heart continued after the breathing ceased, but they did not either control or ascertain the proportion which the vapours of chloroform bore to the inspired air. In Mr. Thomas Wakley's experiments the action of the heart continued after the breathing had ceased; and this was the case in a great number of experiments performed by a Commission which reported to the Society of Emulation of Paris in 1855." This Commission came to the conclusion that "in all instances in which animals are killed by chloroform the action of the heart survives the respiration; but they might have administered chloroform to an equal number of human patients without any one of them being cut off by sudden paralysis of the heart." Evidence, as we all know, is of two kinds—positive and negative; and if three cases of death in the human subject are brought forward which have happened in England or in climates resembling ours in which the heart has ceased beating and the patient has gone on breathing afterwards, then I affirm that these cases are of far more importance to us as practical anæsthetists, and far outweigh the 430 experiments which were performed on dogs, monkeys, and other animals in the tropical heat of India.*

The Commission states that it has searched the records of accidental deaths from chloroform in Great Britain since 1855, and they find that there is not a single death from chloroform recorded in which it was proved that the respiration alone was attended to throughout the inhalation. I also have carefully gone through these same cases, and the conclusion I arrive at is that all these cases were watched with the greatest care on account of the administrator not knowing from which quarter danger might arise, and although it is not specially stated that the respiration was watched, I cannot for one instant doubt that it was well attended to. But to my mind this is a side question altogether, for, from the details of cases quoted a little further on, there can be no doubt that the primary cause of death was cardiac failure, cessation of respiration being secondary. One very important point to note is that in these cases death took place in one instance in 120 seconds, in another in about 60 seconds, and in a third at five minutes from the commencement of inhalation. Now notice how different these times are from those in the fatal cases of the Commission. Not one of the 197 animals died in as short a time as the second case quoted; while the heart of one dog went on struggling for over ten minutes after respiration had ceased, not counting the time of inhalation. There is no doubt that in the human subject, when the breathing stops, artificial respiration is the best proper treatment, and these are the cases which cause the surgeon intense anxiety, but which do not, or very rarely, end fatally; but when sudden and general pallor comes over the face together with sudden loss of pulse, then the patient is dead in ninety-five cases out of a hundred.

It may be objected that in quoting these cases I am going a long period back; but they are typical cases, and they have gone on occurring in much the same way up to the present time, and they will continue so to do as long as chloroform is administered. I have the less hesitation in quoting these particular cases, for in each of them the anæsthetic was given without any inhaler, all forms of which the Commission condemns. Referring to the three fatal cases above mentioned from Snow's work on Anæsthetics, I find :—

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* [The temperature of the Laboratory in which the Commission's experiments were carried out varied from 18° to 24° C.]

CASE 1.—Hannah G——, aged fifteen, of Winlaton, near Newcastle, operation to be performed being evulsion of a toe-nail, a similar operation having been performed under ether a few months previously. On Jan. 28th, 1848, chloroform was administered upon a handkerchief, a drachm being poured on it, patient being seated in a chair. In half a minute Dr. Meggison, the administrator, finding no change in the breathing or alteration of the pulse, tested her sensibility by pinching the cheek, and finding no reflex, told the surgeon he might commence. Patient moved when the incision was made, and more chloroform was about to be given, when the following events happened : lips suddenly became blanched, and she spluttered at the mouth. Cold water was dashed on the face, and the patient laid on the floor. Venesection in the arm and in the jugular vein was performed, but no blood flowed. The entire proceedings from the commencement of inhalation is said to have been two minutes. From the lips becoming suddenly blanched in the above case there is every reason to conclude that the heart was suddenly paralysed. The patient breathed for a little after this, and was able to swallow, though with difficulty. (Dr. Snow's remarks.)

CASE 2.—Patrick C——, age not stated. March, 1848. Disease, fistula. Had taken chloroform once previously. Time of inhalation about one minute. Quantity consumed half a drachm. Lapse of time till death about one minute. Patient on his side, chloroform was administered on a handkerchief or towel. Immediately the operation was commenced the patient became pulseless, his pulse previously being full and natural. Here the period of inhalation was very short, one minute, and the evidence of paralysis of the heart distinct.

CASE 3.—J. V——, aged seventeen. Hôtel Dieu, Lyons, January, 1849. Amputation of finger. Chloroform was dropped upon gauze spread over the face, leaving a free passage for air. At the end of five minutes patient still felt and spoke, at the end of six minutes he became restless and still spoke. One drachm and a half of chloroform had now been given ; pulse regular and good. He now struggled violently, and within a quarter of a minute the pulse at the wrist became imperceptible. The gauze was removed, no pulse anywhere to be felt, and no cardiac sounds audible. Respiration continued, and ceased in half a minute. Means of resuscitation were employed, and respiration reappeared in two minutes' time, and then again gradually ceased. The pulse did not return.

And now we come to the practical conclusions which are generally the first read, and are usually the most interesting parts of the whole paper, no matter on what subject it is written. It is difficult to see how the Committee arrived at its first practical conclusion as to the position of the patient on the back being the best. This position is not so comfortable to the patient as when lying on his side ; any of the anæsthetic fluid accidentally spilt falls on his face instead of on the pillow, and, in fat people especially, the diaphragm does not descend so well owing to the weight of the fat on the abdomen pressing on it. Again, in this position the patient usually swallows all the saliva which is secreted, often in large quantity, and this produces after vomiting; whereas when the patient is on his side the saliva will run out of the corner of his mouth, and consequently cannot irritate the mucous membrane of the stomach. Also, when the patient is lying on his side, the tongue is not liable to fall back and thus impede respiration; in natural sleep and in cases of apoplexy how constantly do we notice that a patient on his back snores heavily and breathes uneasily, but when turned on his side all noise ceases and he breathes quietly. The next conclusion I agree with, provided the patient is placed on his side for the reasons above stated. The principles of the next five paragraphs have been taught in the London schools for years. I can answer for Charing-cross Hospital, where the pupils are systematically instructed in the theoretical and practical details of anæsthetics. In paragraph 33 of the report the Committee state that they performed a large number of operations which are reputed to be particularly dangerous from shock, such as extraction of teeth, evulsion of nails, section of muscles of the eye, &c. These operations were performed in all stages of anæsthesia, and even when the animal was merely stupefied with chloroform; in no case was there anything suggestive of syncope or failure of the heart's action. And yet in Conclusion VIII. we find, "As a rule, no operation should be commenced until the patient is fully under the influence of the anæsthetic, so as to avoid all chance of death from surgical shock or fright." Now, how are we to reconcile these to statements? Is it not apparent that animals do not suffer from surgical shock and cardiac failure, and are in this respect different from human beings. I venture to state that there is no anæsthetist, even of moderate experience, who has not noticed cases of shock from insufficient anæsthesia, due to the surgeon commencing the operation before asking the administrator whether the patient is ready.

Conclusion IX. states: "The administrator should be guided as to the effect entirely by the respiration. His only object, while producing anæsthesia, is to see that the respiration is not interfered with." From the large experience I have had of administering anæsthetics in England during the last thirty years, I feel absolutely certain that if this deduction is acted upon, the number of fatal cases, even now too numerous, will rapidly increase. I maintain that the administrator should watch equally the face, the pulse, and the respiration, sudden pallor of the face being of far more importance than partial interference with the breathing. The precautions mentioned in the remaining paragraphs are usually carried out in the London Hospitals. Time alone will show whether the sanguine future the Commission believe to be in store for chloroform will come to pass if their rules for its administration are carried out.

I remain, Sirs, yours faithfully,

Feb. 3rd, 1890. WOODHOUSE BRAINE.

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## LETTER FROM MR. ROGER WILLIAMS.

### TO THE EDITORS OF "THE LANCET".

SIRS,—Just as modern industrial life is the outcome of the introduction of steam, so modern surgery is based upon the use of anæsthetics. Whatever relates to fatalities during anæsthesia is therefore of the greatest importance to every practical surgeon. With nearly half a century's experience at our back, it is surprising,—in these days of advancing science and greater accuracy in many respects,—to find that even now we have no really reliable information as to the relative frequency of such occurrences. Rough estimates there are in abundance, the value of which may be judged from the fact that they vary, for chloroform, from 1 in 36,500 to 1 in 2,666 administrations.

My object in writing this letter is to point out that a perfectly reliable source of information is now available, so that in future there can be no excuse for ambiguity. For many years past there has been kept at St. Bartholomew's Hospital a most admirable record of the administration of anæsthetics and of the fatalities which have occurred. These have been published from year to year with the annual statistical reports of the hospital. Believing the subject well worth a little trouble, I have tabulated these records for the ten years 1878—1887. During this time chloroform was administered 12,368 times, with 10 deaths (1 in 1,236). During the same period ether was administered 14,581 times, with 3 deaths (1 in 4,860). In 9,072 of these cases ether was preceded by gas; 1 fatal case belongs to this category. In the other 5,509 cases ether alone was given, and 2 deaths occurred.* These facts are very eloquent; they require no lengthy comment. I have long been aware of the greater safety of ether, and have therefore preferred it to chloroform in most cases, notwithstanding its disadvantages in some other respects. I believe this is the goal towards which professional opinion is steadily moving; and I think this movement likely to be most beneficial. Experience at other hospitals leads me to believe that the results obtained at St. Bartholomew's may be accepted as reliable averages. There can be no doubt, in these cases at least, that most of the fatalities occurred in spite of the greatest skill and care being used in the administration of the anæsthetic agents. Such being the case, it is impossible to arrive at any other conclusion than that such occurrences are unavoidable in a certain proportion of cases.

I cannot conclude this letter without emphatic protest against the dictum of the Hyderabad Commission, that such deaths must be ascribed entirely to carelessness on the part of the administrators. Such a statement is opposed to all clinical experience, and it is simply preposterous.

I am, Sirs, yours truly,

Welbeck-Street, W., Jan. 30th, 1890.

W. ROGER WILLIAMS.

## LETTER FROM SURGEON-MAJOR EDWARD LAWRIE.

To the Editors of "The Lancet".

Sirs,—In *The Lancet* of Feb. 8th and 15th, 1890, there are important letters on the relative safety of anæsthetics. Mr. Roger Williams gives certain statistics of the administration of anæsthetics and of the fatalities which have occurred at St. Bartholomew's Hospital, which, he says, may be accepted as reliable averages at the other London hospitals. From these it appears that deaths from chloroform in the London hospitals amount to about 1 in 1,236, and from ether to 1 in 4,860 administrations. The late Mr. Syme's cases and my own form a continuous series of daily administrations* of chloroform in Europe and in India, from 1847 to 1890, without any deaths, and if Mr. Roger Williams's reasoning from statistics is correct, it is logical to infer that the administration neither of chloroform nor of ether, as conducted in London, is as safe as the administration of chloroform on the principles advocated by Mr. Syme.

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* It is right to state that in each of these cases the patient was in an exceedingly feeble and collapsed state before exhibition of the anæsthetic owing to prolonged intestinal obstruction.—W. R. W.

There can be no doubt that there is a flaw somewhere in the London teaching with reference to anæsthetics, and evidence on this point is supplied by the letter of Mr. Woodhouse Braine, who maintains that it is far more important to watch for pallor of the face than for partial interference with the breathing in chloroform administration, having previously stated that pallor of the face is one of the signs that the patient is dead. Mr. Braine writes as if the acceptance of the truth about chloroform would be disastrous to anæsthesia specialists. The reverse of this would be the case, as it is well known that immense numbers of people are deterred from having operations performed on account of the dread of anæsthesia caused by the present position of anæsthetics.

It is for anæsthetists themselves, however, or for the surgeons who employ them, to decide which policy is likely to be more conducive to their own interests; to persevere in announcing publicly that anæsthetics cannot be given safely in London, or to give a fair trial to Syme's principles, which have stood the test of experience, and have been proved by the Hyderabad Commission to rest on a secure physiological foundation. The question of chloroform administration is no longer one of doubt or of opinion, and moreover it is one of common sense. If Hyderabad students can be taught to give chloroform as they do day after day, with guaranteed safety, *a fortiori* it must be a much more easy task to teach London students to do the same.

I am, Sirs, your obedient servant,
EDWARD LAWRIE,
Surgeon-Major.

Hyderabad, Deccan, March 12th, 1890.

## MEDICAL SOCIETY OF LONDON.

An ordinary meeting of this Society was held on February 10th, the President, Dr. Theodore Williams, in the chair.

The President said that so much hung upon the valuable work of the Commission at Hyderabad that the council of the Society had decided to give up an evening to listening to Dr. Lauder Brunton's account of experiments, and to the discussion following thereon.

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[* By daily administrations must be understood several administrations every day.—Ed. L.]

Dr. Brunton commenced by stating that the conclusions arrived at by the Commission had already been published by "*The Lancet*", but he thought that the main value of their work lay in the number of experiments that had been done, which had been recorded graphically, and which would be printed and circulated so that they might be available to all, and the conclusions of the Commission concerning them might be criticised, and, where necessary, corrected. Syme attributed the absence of fatalities in the use of chloroform at Edinburgh to two facts—that very pure chloroform was used, and that plenty of it was given. It occurred to the speaker that one reason of the deaths from chloroform might be the neglect of the second of Syme's dicta; for many of the deaths appeared to be due to shock, and to occur at the beginning of an operation. Syme taught also that in administering chloroform one should watch the respiration and not mind the pulse; and Dr. Lawrie, in the first Hyderabad Commission, instituted a series of experiments to show that the respiration failed before the pulse. In the first Commission 141 animals were used, and they all died of respiratory failure. The second Commission was inaugurated to repeat and extend these experiments, and 430 animals were used with a similar result; in the entire 571 animals the respiration failed before the heart. It might be said that two chief theories were promulgated regarding the action of chloroform: one, which might be called the Edinburgh theory—that chloroform paralysed the respiration, but did not paralyse the heart; and the other, which was chiefly current in London—that chloroform paralysed the heart, and that this was the chief source of danger. It had occurred to Dr. Brunton that the small amount of beer drunk in Edinburgh might lessen the liability to fatty degeneration of the heart and might have something to do with the difference in the mortality. The Committee of the British Medical Association in Glasgow found that chloroform sometimes paralysed the heart, and that this might happen in an unforeseen way which could not be prevented. The experiments of the Commission were directed towards determining the action of chloroform upon the heart, and careful tracings of blood-pressure were taken. An ordinary manometric apparatus was used, the carotid of the animal under chloroform being exposed in the usual way, the distal end being tied and the proximal connected with a mercurial manometer; the tube between the mercury and the manometer being filled with solution of bicarbonate of sodium. Instead of using a quick recording drum, which would give an immensely long tracing, a slow drum was used revolving only once in half an hour. This, though it recorded the effect of respiration only, showed nevertheless sufficient for practical purposes. But in order to register any particularly interesting variations found on the slow drum with more accuracy, the glass tube connecting the artery with the manometer was furnished with a branch, and this led to a second manometer and a quick drum. With this apparatus, by simply shifting a clamp, a "Sample," so to speak, of the cardiac action could be taken on the quick drum at any interesting moment. To avoid errors from inertia the quick drum was worked in connection with a Fick's manometer. They found that the difference between chloroform and ether, so far as their effects on blood-pressure was concerned, was just what Claude Bernard had pointed out years ago, that they both acted in a similar manner, but that chloroform was the more powerful of the two. Under chloroform the pressure sank comparatively quickly, and on its stoppage and the adoption of artificial respiration it gradually rose again. If ether was used there was a much slower fall of pressure, and artificial respiration produced a similar rise. If air strongly charged with chloroform were used, a sudden fall of pressure was observed. It then behoved them to inquire how deaths during the administration of chloroform were likely to occur, and how they were to be prevented. They investigated the direct action of chloroform upon the heart. It was well known that if chloroform were injected into ordinary striated muscle it became hard like a board, but they found that injection of chloroform into the veins did not produce a distinct action on the cardiac muscle fibre,—at any rate, not in anything like the degree they had anticipated. The injection of both chloroform and ether into the veins was found to lower the blood-pressure, but ether had a less effect than chloroform. It was found, however, that ether usually caused clotting of blood in the heart, and thus led to a fatal result. When ether and chloroform were injected into the trachea they both produced a rapid fall of blood-pressure. It should be remembered, however, that the action of chloroform in these two ways,—by injection into the veins and into the trachea,—represented its toxic rather than its therapeutic effects. If the vapour of chloroform were driven into the trachea after the stoppage of respiration, the heart became paralysed; but when administered by simple inhalation alone, when the respiration stopped an excess of chloroform was prevented from getting into the circulation, and the heart in that case was not paralysed. But there were certain points about the matter which required fuller consideration. An animal dying from paralysis of respiration might, after the stoppage of regular breathing, make one or two violent gasps, and these might bring in enough chloroform to stop the heart's action. There was some risk of this happening, but this risk only occurred if the chloroform vapour was drawn in when the animal was in a state of asphyxia. A tracing taken from an animal in this condition was found to be identical with one produced by the Committee of the British Medical Association. There was a drop in the respiratory curve, followed by slow action of the pulse, and then a gradual rise again. This slow pulse was due to vagus irritation, and such a tracing could be made at will by inducing simple asphyxia, either by closing the dog's nostrils, or, if a canula were in the trachea, by stopping the end of the canula, or by making the dog breathe pure carbonic acid gas. Such a condition was never produced by the administration of chloroform with plenty of air, but it always occurred during accidental asphyxia. Dr. Brunton had been much puzzled as to how the Glasgow Committee had produced this tracing, and he suggested an explanation. If, with a canula in the trachea, artificial respiration were being performed, a small quantity of blood might run down into the bronchi and clot. It might there form a valvular plug, and by moving up and down produce an intermittent asphyxia and give rise to such a tracing. Several experiments were tried on the effect of shock, but no marked symptom of that condition could be produced. It was true that no experiments had been done without ether or chloroform, but they concluded that such experiments were not only painful but unnecessary. They first put an animal under chloroform and extracted several teeth, but no symptom of shock followed; they then tried laying hold of the skin of the anus and pulling and snipping it, imitating operation for piles, but with no result. The abdomen was then opened, and a piece of intestine pulled out and ligatured; a blow was struck upon the testicles as the animal was coming round, but the effects were not so marked as would have been imagined. As to position, when the animal was put upright there was a distinct fall in blood-pressure, which became normal when the horizontal posture was resumed; elevation of the legs was found to raise the pressure. With the animal in the upright position, striking the abdomen or extracting teeth produced no shock. The effects of dress were tried on female monkeys; a plaster-of-Paris jacket was put on to imitate stays, and a tight bandage round the abdomen to resemble the band of a petticoat. Under both these disadvantages the animals seemed to die very quickly indeed. Phosphorus was administered to induce fatty degeneration, and in some the heart seemed to stop more quickly than in healthy animals, but the effects were not marked. Their main conclusions were that chloroform and ether acted in the same way, both paralysing the respiratory centre before the heart; if pushed after the respiration had ceased, the heart would be paralysed. Chloroform was more potent than ether, which latter could not be used in very hot countries, and the chloroform should be given with plenty of air, for suffocation and chloroform formed a deadly combination. The deaths from chloroform were due to asphyxia and resulted from imperfect observation of the respiration. During the prosecution of the research they had a large number of accidental deaths, but all these were due to inattention to the respiratory condition. The pulse did not begin to fail till after the respiration, and if the respiration were not attended to till after the pulse had failed it was probably too late. If attempts were made to pay attention to both the respiration and the pulse, neither perhaps would be observed well, and hence it was advisable to keep the attention fixed on the respiration alone. Dr. Brunton concluded by saying that we owed a great deal to the Nizam, who not only caused the research to be undertaken, but had generously consented to publish it, so that a complete set of the photographic tracings would be presented to all the principal medical libraries in the world.

In the discussion which followed, Mr. Bailey dealt with the clinical side only. In the early days of chloroform he thought it was administered as well in London as in the north, but many deaths occurred which were considered to be due to failure of cardiac action. The plan of administering nitrous oxide before ether did away with its disadvantages, and as perfect an anæsthesia could be obtained with ether as with chloroform, and the death-rate from the former was smaller. He found it quite easy to watch not only the respiration, but also the pulse and the pupil, and the latter, he thought, gave most valuable indications as to the patient's condition.

Mr. Braine said that in fatal cases under chloroform the patient would be going on well, then a little more chloroform would be given, when a pallor of the cheeks would be noticed and no pulse would be felt, though the patient breathed on. He thought that one case so observed in the human being was more valuable than thousands of experiments on animals.

Dr. Sansom agreed that it was most valuable to have these experiments on blood-pressure reduced to manometric certainty. The question was what were the teachings of experiments on animals, and how did they bear on the practice of the present day? Years ago careful experiments were performed both in England and abroad, which led to the same conclusions that the Hyderabad Commission had shown more elaborately, namely, that the heart in animals was an *ultimum moriens.* But animals varied very much in their reactions to anæsthetics even in the same species, and he thought that we could not argue direct from animals to man, whom the influence of emotion placed in an entirely different category. While there was a fair amount of uniformity in the necropsy records from animals, in man this was not so, for in some cases all chambers of the heart were found full of blood, which pointed strongly to the idea that death took place from paralysis of the heart rather than of the respiration. His general conclusion was that, though experiments were valuable, they ought not to be accepted as solving the question, and while practical anæsthetists valued these experiments at their proper worth, they could not prevent the possibility of syncope occurring.

Mr. Carter referred to a case which occurred years ago when he was giving an anæsthetic, and the patient died. He could not remember the state of her respiration, but saw nothing to arouse suspicion till he noticed the pallor of the countenance. His own opinion was strongly in favor of chloroform as opposed to ether for ophthalmic operations. Ether was attended by more struggling and consequent venous congestion, and the liability to hæmorrhage from the choroid was therefore greater.

Dr. Routh advocated the use of methylene as an anæsthetic. He had seen apparent death follow the administration of an anæsthetic in six cases ; in all the respiration seemed to fail as much as the pulse, and they were all restored by turning them topsy-turvy. He had likewise seen a fatal case from ether administration. He thought that an apparatus for the registration of chest movements would be valuable.

Dr. Day had administered methylene 1300 times without an accident. He had given it in cases of weak and dilated heart and in mitral stenosis without bad effect. He quoted from Sir Spencer Wells's work to illustrate its value.

Dr. Hewitt had never known respiration to fail while the pulse was good or fair at the wrist, and he had seen two or three cases in which the pulse had given the first sign of danger.

Dr. Brunton, in reply, said that there were certain differences of opinion as to the relative advantages of chloroform and ether. He had found the respiration stop in animals when the pulse was quite good at what corresponded to the wrist. Their experiments with ether were done under difficulties owing to the high temperature in Hyderabad ; they had almost to cut off the air before they got anæsthesia with ether. After poisoning from much diluted chloroform there was a great difficulty in bringing the animal round, but there was not this difficulty with a stronger dose ; the reason was that in the former case it was difficult to say when the animal had stopped breathing. He felt sure that the truth about the question would come out later when the whole investigation was published, and when others would have an equal opportunity with themselves of studying the work that had been done.

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## ANNOTATION FROM *THE LANCET*, FEBRUARY 15TH, 1890.

Dr. Lauder Brunton delivered a very interesting address on his work at Hyderabad at the Medical Society on Monday evening. Describing the methods he employed, now pretty familiar to those who have pursued physiological research, he passed round a number of tracings illustrative of the fall of blood-pressure under chloroform, &c. Dr. Brunton reiterated in the main the conclusions which have already appeared in our columns, but supplemented them by details of work which could hardly find a place in the formal report. The discussion to which Dr. Brunton's singularly lucid descriptions gave rise tended to indicate conclusions not altogether in harmony with the Hyderabad Commission's results. While admitting the force of Dr. Brunton's arguments as applied to the lower animals, the practical anæsthetists present deprecated the extension of conclusions from the lower animals to man, unless positive evidence of uniformity of behaviour of chloroform towards men and beasts were adducible. It was further pointed out that the evidence produced was, so far as the action of the drug upon the heart went, wholly negative, and that the clinical observations of Snow, Clover, and living anæsthetists were opposed to the Commission's contention that chloroform kills through the failure of respiration, and not by primary heart failure. While conceding the obvious and great value of experiments made upon the lower animals to elucidate conditions prevailing in man, one of the speakers pointed out that considerable divergence in reaction towards chloroform existed in them, and this was an additional reason for not relying too much in the present discussion upon the negative evidence Dr. Brunton advanced. None will seek to diminish the great value, both scientifically and practically, of Dr. Brunton's painstaking researches, and it must be accepted as a sign of respect to him that so eager and lively a discussion was elicited by his description of his part in the work of the Hyderabad Chloroform Commission.

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## LETTER FROM Dr. DUDLEY BUXTON.

### To the Editors of "The Lancet."

Sirs,—The publication of the report of the Second Hyderabad Chloroform Commission and Dr. Lauder Brunton's explanatory remarks given to the Medical Society afford very many points for careful study. It is unnecessary for me to dwell upon the debt we owe to Surgeon-Major Lawrie, to the public-spirited Nizam of Hyderabad, or to Dr. Brunton and the other members of the Commission. Dr. Brunton's work has always been so good, so thorough, and so earnest that I believe all who are interested in this most important question—Can chloroform be given safely, if given properly?—looked forward with the utmost interest to the feast of reason which he, alike with the other members of the Commission, was to place before us. Now that we have got it, are we happy? Dispassionate candour compels me to reply that I at least have been carried no farther, although setting main issues aside, I would add no one can read the suggestive report without gleaning much that is valuable and much that is instructive. Of the many points involved, I can crave your indulgence for reference only to a few. In the first place, I find no attempt is made to bridge over the great hiatus betwixt experiment upon the lower animals and the daily experiments made on man. Possibly this is to come. Again, I am disappointed to learn no authoritative statement as to whether dogs, monkeys, &c., are liable to syncope under any conditions; personally, I believe if they are so the occurrence must be most rare. Comparing the statements concerning the lower animals with one's own experience among human beings, a wide discrepancy occurs; for every grade of heart weakness finds a record in the note book of every observant anæsthetist, provided his hospital experience is large; nor can the bulk of such cases be attributed either to primary failure of respiration or careless administration. No doubt maladministration may account for some deaths under chloroform, but not all, since such fatalities have occurred under the skilled hands of men whose knowledge and experience were great and whose reputation was at stake. Again, we have long acknowledged the danger of death from asphyxia occurring during chloroformisation, and Snow pointed out long ago how over-dosage would, by paralysing the medullary centres, bring about death ; but our experience—and it must be conceded that a daily round of experiment and observation made upon human beings and conducted through many years gives practical anæsthetists a right to a moderately authoritative judgment in the matter—repeated again and again, tells us that in a certain number of persons the pulse flags, loses tension, and fails, even though respiration remains unimpaired until with cardiac failure respiratory rhythm ceases. That deaths occur in the initial stage of chloroform inhalation is commonly reported ; but if it be contended that such arise from fear, as in the oft-quoted case of Simpson, confusion becomes worse confounded, for patients exposed to the same terrors from nitrous oxide gas, which has been administered many millions of times, and from ether, do not die in the same way ; so that we are forced to believe chloroform, however it does it, does kill in a way peculiar to itself, which no forethought can anticipate and no care or skill can obviate. Again, we must remember that temperature has much to say to shock and heart failure, and it may not be irrelevant to suggest that the Tropics in this way may contrast favourably with temperate zones. Dealing with the comparative safety of chloroform and ether, I think we may be pardoned if we decline to accept as proven the conclusions arrived at by the Hyderabad Commission. In the first place Dr. Brunton told us that his ether was impure ; secondly, that in the climate of India it is impossible to obtain anæsthesia by ether and maintain it unless air be so rigidly excluded that suffocation occurs. Under such circumstances, ether is no doubt a deadly anæsthetic. In temperate countries ether is not given in this way, and no asphyxial phenomena are present. To describe ether narcosis as semi-anæsthesia is to prove that the modern methods of giving ether are unknown to the writers, and to invalidate their strictures upon that safe and most valuable anæsthetic. In conclusion, I would add that the experiment undertaken by the Commission to elucidate shock under anæsthesia is again totally opposed to our clinical experience. Were the conclusions advanced to be accepted by us, we should find it hard to explain the fluctuations in the pulse, the differences in respiratory rhythm which occur in laparotomies when the intestines are handled, the pedicle of an ovarian cyst is dragged upon, or a bladder is seized and held while punctured in suprapubic cystotomy. The division into "chloroformists" and "etherists" is irrational and harmful ; every anæsthetist learns sooner or later that every case is *sui generis* and a law to itself, and for each must he decide between not only chloroform and ether but between them and the many other methods of mixed narcosis with which we are now familiar. Without fear or dread must he be prepared to give one or the other anæsthetic, but he must be keenly alive to *all* the possible contingencies of each, and not live in a fool's paradise that if he only obeys certain rules and directions he and his patient are safe and he may administer his anæsthetic " with perfect ease and absolute safety so as to do good without the risk of evil."

I am, Sirs, yours, faithfully,
DUDLEY W. BUXTON,
Anæsthetist to University College Hospital.

Mortimer-Street, W., Feb. 1890.

# LETTER FROM DR. SHAND.

## TO THE EDITORS OF "THE LANCET."

SIRS,—This being a most important subject, I feel morally called on, as one who has had nearly fifty years' experience in practice, to express my dissent from two at least of the conclusions arrived at by the Chloroform Commission. I may premise that I used ether first with satisfaction till chloroform appeared, and saw it extensively employed in 1848 on the wounded from the various engagements on the advance of the Prussian troops to the relief of Radstadt and the restoration of the Grand Duke of Baden. So that I was the first English doctor (and from Scotland too) who saw chloroform used in gunshot wounds. With regard to the second conclusion, namely, the necessity of recumbent position in inhaling chloroform, I beg to state that two of my most satisfactory operations were performed many years ago under chloroform in the sitting position. One was a gunshot wound of the right hand, which so smashed all the metacarpal bones that it was only possible to preserve the thumb and forefinger. The gentleman is in London at present, and could exhibit the *claw*, as he terms it, with which he writes and shoots as of old. I saw him soon after the accident, and with the assistance of a butler placed him in an arm-chair, chloroformed him, and performed the operation. The second was a case of incarcerated inguinal hernia of the left side. I fixed the patient in a chair with the counterpane of his bed and gave him the chloroform, while I sat on a chair facing him close to the window with my back to the light. The cure was rapid and perfect, and the patient required no truss. He was well in four weeks or less. I give you these two cases in respect to position. A large percentage of people respire more freely in that position than in the recumbent one; and although I do not think the heart's action is to be neglected, yet even for its benefit the respiration is the key. My experience of pure alcoholic chloroform, administered by an expert, is that it is the most manageable, safe, and effective of anæsthetics, although I quite agree with Sir Spencer Wells, that, provided the administrator is an expert in his own choice, any of at least three will answer equally well. The next material point is as to administering morphia, either hypodermically or otherwise, before chloroform. A dose of morphia often appears harmless, and obtains a good response. I have extracted a pebble from the hip, the patient resting on his face in bed, and succeeded well enough, though, as might be anticipated, I had to restore respiration in the middle of the operation by pressure of the ribs at an open window. I have administered sufficient chloroform several times to keep the patient asleep for about twenty-four hours right off, and without accident.

P.S.—Since writing the above I notice that one of your correspondents mentions two important points in last week's issue, one of which is the advantage of placing the head on one side during the inhalation of the drug, to allow the saliva to flow out of the corner of the mouth. It also almost prevents the falling back of the tongue over the epiglottis. This arrangement I have frequently alluded to and acted upon with satisfactory results.

I remain, Sirs, yours faithfully,

JOHN SHAND, M.D., F.R.C.P., ED.

Edinburgh, January, 1890.

# LETTER FROM MR. EWEN J. MACLEAN.

## TO THE EDITORS OF "THE LANCET."

SIRS,—The following notes of a case in one's experience of the use of chloroform may be of interest at the present time. J. G , aged fifty-nine, army pensioner ; has seen a good deal of foreign service : had malaria. Operation by colleague : evulsion of the right great toe-nail. Patient had been low-spirited for some time. Bowels regular. Had no food for four hours previously to operation. Chloroform administered by myself on an ordinary soft bed-room towel, folded cone fashion, after the heart had been examined as a matter of form and found normal. The head was kept low. It was a considerable time before the anæsthetic took any visible effect, and the patient was very quiet both in speech and movement and took deep breaths freely. Somewhat marked congestion of the external jugular and temporal veins was noted. Three times the operator tested the seat of operation after the conjunctival reflex appeared abolished and muscular relaxation present, but found sensibility to pain still retained. The fourth time, however, there was no response, and the operation was proceeded with. In the course of five minutes or so, when the toe was being sponged, while the pulse remained steady, though a trifle rapid, the breathing became quicker and somewhat shallow. The administration of chloroform was at once stopped and the cloth put away to a safe distance. The breathing, however, became increasingly shallow and soon stopped entirely. In the mean time the lips and face generally had become distressingly livid and the pupils widely dilated. The pulse, however, kept going, though its character or frequency cannot be reliably noted from the fact that artificial respiration was at once resorted to. Silvester's method was employed, the tongue being drawn forwards with forceps by my colleague. The windows of the room were widely opened to admit air freely. After about two minutes of this treatment the artificial respiration was stopped, to see if auto-respiration would be re-established, but still no breathing. There was no time to examine the characters of the heart or pulse. Cold water was next dashed in abundance over the chest and throat, and this undoubtedly effected the turning point in the case, as, after a few more passes of the Silvester method, auto-respiration returned—at first very shallow, but rapidly improving. The recovery seemed accelerated by the application of flannels wrung out of boiling water to the præcordia. The patient was next got comfortably into bed with hot-water bottles to feet and flanks and brandy and ether were administered. He recovered without any apparent ill-effects, and next day, when told of our anxiety as to his welfare on the previous day, stated that it was the seventh time in his varied experiences he had narrowly escaped with his life. This case, amongst other things, impresses on one :—(1) The necessity of watching with special care those cases where anæsthesia is difficult to establish. (2) The importance of noting the character and frequency of the respiration. (3) The fact that chloroform paralyses the respiratory before the cardiac centre. (4) The value in these cases of Silvester's methods of artificial respiration. (5) The great value of cold-water affusion in recovering auto-respiration. (6) The moral duty of being thoroughly well acquainted with the treatment of emergencies incident to chloroform administration.

I am, Sirs, yours very truly,

EWEN J. MACLEAN, M.B.

Bristol, February 1890.

## LETTER FROM THE GLASGOW CHLOROFORM COMMITTEE.

To the Editors of "The Lancet."

Sirs,—The Hyderabad Report, which appeared in "*The Lancet*" of January 18th, naturally attracted our attention and called for remark, especially as it seemed to traverse certain of our conclusions. On carefully reading the report, it appeared to us that, so far as the facts are concerned, it generally confirmed our own observations as to the action of chloroform both on respiration and on the heart. Some of the inferences, however, are opposed to ours; but they are also opposed, as we believe, to the facts stated in the report itself. We therefore propose to criticise the report, and we would have done so immediately after its publication but for a request communicated to us that we should wait for the publication of the tracings on which the report is largely based. *We at once agreed to postpone our remarks*,* and only make this communication lest our silence should be misinterpreted.

We are, Sirs, yours faithfully,
JOHN G. McKENDRICK.
JOSEPH COATS.
DAVID NEWMAN.

## LETTER FROM Mr. FREDERIC HEWITT.

To the Editors of "The Lancet."

Sirs,—Although wishing that space would permit me to express my keen appreciation of the work done by the Commission, I feel that, as there is so much to be said on this important subject, I must at once proceed to the consideration of the questions at issue.

In the first place I would ask what is the true value of the physiological fact that, when chloroform is administered in toxic doses to the lower animals, respiration ceases before cardiac action? Have we any clinical evidence to prove that, *under similar circumstances*, this sequence of events is not met with in human beings? In most, if not in all, of the rapidly fatal cases which have occurred under chloroform, it has, for obvious reasons, been a matter of extreme difficulty to say at what particular moment the heart ceased to beat. Failure of the pulse has often been taken to mean stoppage of the heart, but without sufficient grounds. If it were possible to make a series of observations upon human beings with that accuracy which is attainable when conducting experiments upon lower animals, I should not be surprised to learn that, when chloroform causes death solely by reason of its toxic properties, the same sequence of events as that observed by the Hyderabad Commission invariably occurs. But our knowledge concerning the action of the heart under anæsthetics is almost entirely dependent upon observations on the pulse; and it would seem that confusion has frequently arisen in conse-

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[* In spite of this statement the Glasgow Committee published their remarks one week before "the publication of the tracings on which the report is largely based."]

quence of failure of the pulse having been taken to mean that the heart has "suddenly ceased." I am here only referring to those cases in which chloroform itself would seem to have been the cause of death; and in such cases, which are usually rapid in their course, I do not think the clinical evidence we possess is sufficient to disprove the contentions of the Commission. But this, I submit, is the least important part of the subject. Even though we admit the fact that, when chloroform itself is the direct cause of death, respiration ceases before the action of the heart, we are confronted by a question which seems to me far more important than that upon which the Commission has laid so much stress. When fatalities occur during the administration of chloroform, are those fatalities invariably caused by the direct toxic effects of the drug? In other words, have we not conclusive evidence to show that, in man, deaths under chloroform sometimes arise by reflex cardiac failure which is only indirectly due to the anæsthetic? I cannot regard the experiments of the Commission in this direction as rendering such a position untenable; for cases have come under my own observation in which symptoms of cardiac depression obviously of reflex origin have arisen. Whether it be the low vascular tension of chloroform narcosis or other conditions I know not; but I feel sure that there is something that renders the human heart under chloroform susceptible to impulses which are utterly inoperative under ether. I am inclined to the belief that the performance of many operations under chloroform is attended by considerable risk from this quarter; and I am by no means satisfied that cardiac inhibition is less likely to be produced during profound than during imperfect anæsthesia under chloroform. Not long ago I administered chloroform, by means of Junker's apparatus, to a patient of about thirty-five years of age, whose general health was good. Anæsthesia was produced in from eight to ten minutes, and was characterised by muscular flaccidity abolition of lid-reflex, and slight stertor. There were two stages in the operation about to be performed, the first of which consisted in placing a temporary ligature round the carotid artery in the neck. Whilst the artery was being exposed for this purpose the pulse became extremely feeble, the face pale, and respiration shallow, and the operator had some difficulty in recognising the carotid artery by reason of its extraordinary diminution in size. The head was lowered. After three or four compressions of the chest the pulse improved, and, as rigidity and lid-reflex soon reappeared, I was obliged to continue the administration, having recourse to ether for the remainder of the operation, which was successfully performed. The day after the operation, whilst the wound over the carotid was being examined, the patient's face suddenly became pale, the artery contracted as on the previous occasion, the eyes were observed to turn upwards and the muscles of the jaw to twitch, and for a few seconds unconsciousness was present. Now in this case, when the first attack of syncope occurred, the patient was thoroughly anæsthetised by chloroform; whilst the quick return of muscular rigidity and of lid-reflex proves that the anæsthetic was in no way to blame as a *direct* cause of the symptoms. Cases of this kind are, I believe, by no means uncommon, and they would seem to point to the conclusion that reflex syncope may undoubtedly arise under chloroform even when the anæsthesia is profound. Apart from anæsthetics, some persons are, as is well known, more prone to syncope than others, and this would seem to be so with regard to patients under chloroform. How can we compare the patient above referred to, to the pariah dog? Would the latter be likely to be attacked with syncope by manipulating its carotid? I gather from the report of the Commission that syncope, should it arise, is a safeguard against chloroform poisoning rather than a condition involving much danger to life; but we cannot look upon it in this light when we meet with it upon the operating table.

In conclusion, I would say a few words concerning the indications afforded by the pulse during chloroform administration. There is much evidence to show that, in whatever way death occurs during chloroform narcosis, the pulse, if carefully watched, usually gives warning of the approach of danger before respiration has become seriously affected. In those cases in which cardiac depression is only indirectly due to the chloroform—such, for example, as the case I have related—the initial symptoms are obviously cardiac in origin, and are hence to be detected by alteration in the force and frequency of the pulse. In those cases, too, in which the symptoms are indisputably due to an overdose of chloroform—such, for example, as the cases reported by the Commission—the pulse will, in obedience to the fall of vascular tension (which, as the Commission admits, precedes stoppage of respiration), give indications of the most important character. If the Commission could prove that, when chloroform is administered in toxic doses, respiration invariably ceases *whilst* the radial pulse is practically unaltered in quality, we should begin to look upon chloroform as a respiratory poison only; but these are not the facts, so far as I understand. I cannot avoid the conviction that the Hyderabad Commission have incurred a grave responsibility in eulogising chloroform as an anæsthetic for general purposes, and in recommending administrators to disregard the indications afforded by the pulse. As I have before ventured to point out in these columns, we should consider the inexperienced rather than the experienced in recommending an anæsthetic. I have lately read the records of every fatal case reported by "*The Lancet*" and "*British Medical Journal*" as having occurred under anæsthetics in the British Isles from 1880 to 1889 inclusive, and I find that out of a total of 130 chloroform deaths no less than fifty-four took place in connection with minor surgical operations, most of which were doubtless conducted with somewhat less caution than would have been employed in more critical cases. Are we to advise the use of chloroform (which Dr. Lauder Brunton admits to be a most powerful drug) to recently qualified men, who have perhaps never employed it before? I confess I cannot regard it as advisable to permit those with but little experience to administer chloroform to patients coming into the surgeries of hospitals with a dislocated shoulder or a lacerated finger, yet this course is one which the Commission appear to countenance. Even though we accept the facts so ably put before us by the Hyderabad Commission, we are, I would submit, in no way justified in agreeing with the practical conclusions at which the Commission have arrived.

I am, Sirs, yours obediently,

FREDERIC HEWITT,

Instructor in and Lecturer on Anæsthetics
at the London Hospital, &c.

George-Street Hanover-Square, W., Feb. 24th, 1890.

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[It is to be observed that when the pulse failed Mr. Hewitt employed artificial respiration to restore it.]

# LETTER FROM Dr. CHARLES E. SHEPPARD.

### To the Editors of "The Lancet."

Sirs,—The importance of the issues at stake seems to render it incumbent upon everyone who has special opportunities for observing the action of anæsthetic agents to record his opinion concerning the results of the Hyderabad Commission. Many of the conclusions arrived at are of the utmost importance, and our best thanks are due to the Commission for bringing these prominently under notice. I allude more particularly to the influence of asphyxial conditions, the previous exhibition of certain drugs, hæmorrhage in considerable degree, and the position of the animal anæsthetised. On the other hand, there are many points for the final settlement of which we had all looked forward with the greatest interest to the experiments of the Commission, but in which we are, if anything, in a more uncertain position than before.

In the first place, it is unfortunate that all the evidence as regards the occurrence of primary syncope is negative, and one cannot help feeling that, even were the instances considerably multiplied, there still would be no certainty that the next experiment might not prove the exception to the rule. And if we are not justified in arriving at a dogmatic conclusion in the case of the lower animals, how much less are we warranted in applying the same conclusion to the human subject. The records of clinical experience, in fact, stand confronted with the results of laboratory experiment, and I think that every one will allow more weight to one positive instance occurring in the former than even to thousands of negative instances in the latter. It is well known that many of the cases which have suddenly terminated fatally, or shown signs of serious cardiac failure during the administration of chloroform, come under a certain category, including forcible dilatation of the sphincter ani and other operations which I need not here specify. Other fatal results have occurred during the performance of trivial operations where the action of chloroform has admittedly not been carried to complete narcosis. Now, although there may be some difficulty in attributing the precise cause in each individual case, whether an overdose of the drug suddenly applied in order to check movement, or whether a reflex inhibition of the heart in consequence of incomplete anæsthesia, there can be no doubt as to the symptoms observed, *viz.*, sudden pallor and failure of pulse, almost coincident with the commencement of operation, and in many cases absolute failure of all means of resuscitation, although promptly applied. Now, on referring to the experiments made upon the lower animals in this connection, we find that, although all the operations associated with shock and cardiac failure were performed in all stages of chloroform administration, yet there was nothing beyond a slight variation in blood-pressure to indicate anything approaching syncope or cardiac failure. Evidently, therefore, in this matter we stand face to face with a serious dissimilarity between the conditions as occurring in the human subject and in the lower animals respectively, and yet, on turning to the practical conclusions deduced from the experiments of the Commission, we find it recommended that "as a rule, no operation should be commenced until the patient is fully under the anæsthetic, so as to avoid all chance of death from surgical shock or fright." With the conclusion I think most chloroformists will agree, but not with the method of arriving at it ; and I maintain that if we followed the indications furnished by laboratory experiments in this instance we should be led into grievous error.

On making a general survey of the experiments as reported we cannot help being struck by one very important fact, *viz.*, the uniformity with which the various phenomena presented themselves in each series, even in those purposely complicated for special objects. In other words, the percentage of typical cases is very large, and contrasts markedly with what is observed in the human subject, where typical form is comparatively uncommon, but is nevertheless, when it occurs, recognised as the normal and made the point of departure for the consideration of all others. It is chiefly on this account that I feel I must disagree with the statement of the Commission that, if the rules laid down be followed, "chloroform may be given in any case requiring an operation with perfect ease and absolute safety." The public, who are our judges, if anything goes amiss, will—nay, already have supplied the words "by any one, experienced or inexperienced," and this idea, if allowed to gain acceptance, will, I am afraid, be fraught with disaster. Even assuming for the moment that primary cardiac failure never occurs in the human subject, cases are continually presenting themselves where the respiratory signs need the closest watchfulness. Clinical experience shows us that shallow breathing occurs at the two ends of the scale. On the one hand, when the subject is deeply under the influence of chloroform; on the other, when he is entering or emerging from its influence. In our typical cases the interval between the two is well marked, and the two conditions are easily distinguishable ; but in others, not at all infrequent, the interval is so small and so slightly marked that it may be bridged over by a minute dose of chloroform, or even by a mere alteration of position in the patient. It is here that even the experienced administrator might be led into a fatal error if he trusted entirely to the ill-defined signs furnished by the respiratory function, and it is only by a careful attention to the important indications yielded by the pulse, the pupil, the conjunctival reflex, the colour, and temperature of the skin, that he is able to conduct his patient safely through a truly perilous journey. I cannot conclude this letter without heartily endorsing every word in Dr. Hewitt's valuable letter on the same subject. I have myself notes of at least two cases in which, during complete and satisfactory chloroform narcosis and without hæmorrhage of any importance, there was a rapid fall in the volume and tension of the pulse, attended with blanching of the lips and coldness of the surface, without any preliminary alteration in the depth or frequency of respiration. Whatever the exact cause may be, there can be no doubt that during the administration of chloroform we must always be prepared for such an occurrence, whereas with ether we may practically disregard its possibility. The duty of the anæsthetist during the performance of a surgical operation on the human subject must be regarded as a highly complicated one, and I am of opinion that the most reliable administrator is the one who, automatically, as it were, is at all times in full possession of the general condition of his patient, keenly alive to all the indications presented to him, however trivial, and at once prepared to regulate his procedure accordingly.

I am, Sirs, yours obediently,
CHARLES E. SHEPPARD.

Second Chloroformist to the Middlesex Hospital ;
Anæsthetist to Guy's Hospital (Dental School).

Welbeck-Street, Cavendish Square, W., March 4th, 1890.

## ANNOTATION FROM *THE LANCET*, MARCH 22ND, 1890.

It is rather extraordinary to observe what an outcry has lately been raised by a number of our lay contemporaries against the experiments upon the effect of tight-lacing as a cause of death during the administration of anæsthetics which were made by the Chloroform Commission in Hyderabad, and mentioned in an address given at the Medical Society by Dr. Lauder Brunton. In some papers those experiments have been quoted as examples of the infliction of pain upon sensitive animals to an extent that is "horrifying," and have been stigmatised as "wanton cruelty." Some of the papers appear to have thought also that Dr. Lauder Brunton's observation that the experiments were made on female monkeys as being more like women than dogs referred to their mental and not to their physical characters, whereas it is obvious that the observation had reference only to the shape of the body as influencing the respiration, and in shape the body of the monkey more nearly resembles the human being than does that of the dog. Female monkeys were chosen in preference to males on account of the possibility that a difference might exist between the amount of thoracic and abdominal breathing in male and female monkeys similar to that which exists in women as compared with men. It is hard to see how these experiments can be looked upon as cruel, inasmuch as there was absolutely no pain inflicted, the experiments simply consisting in applying to monkeys *under chloroform* an amount of compression of the chest and abdomen which is self-inflicted daily by many thousands of women in Great Britain without anæsthetics. The objection has also been raised that the experiments were needless, as the effects of tight-lacing had been demonstrated over and over again; but it seems to have been forgotten that the experiments of the Commission were not made in regard to the effect of tight-lacing in general, but to the effect of tight-lacing as a factor in producing death during the administration of chloroform. Theoretically, no doubt, it ought to be easy to predict that tight-lacing would aid in the production of death during the administration of chloroform; but it is very easy to say, after the experiments of the Commission have shown this to be the case, that the results they obtained were well known before, and that no good has been gained by their experiments upon the subject. But practically the dangers of tight-lacing have not been properly appreciated, inasmuch as an unfortunate death occurred in Edinburgh from nitrous oxide administered for the extraction of a tooth, at the beginning of October last, shortly before the Commission began its labours. In this case there seemed to be reason to believe that tight-lacing had contributed in no small measure to the unfortunate result, and it was, indeed, this lamentable accident which led the Hyderabad Commission to make their experiments. These experiments will have served their purpose if they lead to the general observance of the rule which the Commission lays down in the third section of its conclusions, viz.: "To ensure absolute freedom of respiration, tight clothing of every kind, either on the neck, chest, or abdomen, is to be strictly avoided; and no assistants or by-standers should be allowed to exert pressure on any part of the patient's thorax or abdomen, even though the patient should be struggling violently. If struggling does occur it is always possible to hold the patient down by pressure on the shoulders, pelvis, or legs without doing anything which can by any possibility interfere with the free movements of respiration." The outcry, foolish though it may have been, against the experiments just mentioned may not be altogether without its use if it leads to a general recognition of the dangers of tight-lacing or tight clothing generally in anyone who is about to take an anæsthetic.

## LEADING ARTICLE.

" *The Lancet*," *29th March* 1890.

Professor H. C. Wood, conjointly with Dr. Hare, has communicated a paper upon the physiological action of chloroform to the American *Medical News* of Feb. 22nd. The authors criticise the work of the Hyderabad Commission, and are led to adopt very different views concerning the action exerted by chloroform upon the heart. Moreover, they take exception to an observation made by us in *The Lancet* of Jan. 18th, p. 139. We said, in speaking of the Hyderabad Commission: "The practical outcome of the research would appear to be that deaths are not inevitable. They are therefore preventable, and by due care in its administration they may be with certainty avoided." Professor Wood and his collaborator do not appear to recognise that in using the above words we were expressing the conclusions of the Commission rather than our own. We were careful to add: "The conclusions of the Commission are sweeping, and without abundant evidence cannot be accepted." We have spoken with no uncertain sound from time to time concerning the dangers attending the use of chloroform—dangers which unquestionably, in temperate climes, manifest themselves through the heart. A careful perusal of the report of the Commission will, we think, amply justify us when we assert the outcome of its teaching is that heart failure does not occur, and deaths are, if ordinary precautions are adopted, quite preventable. We may, however, quote the final words of the Commission in confirmation of this. It says (*The Lancet*, Jan. 18th, p. 159): "The Commission has no doubt whatever that if the above rules be followed, chloroform may be given in any case requiring an operation with perfect ease and absolute safety, so as to do good without the risk of evil." The rules, we may remark, are simply those which every competent chloroformist has, since the days of the English Chloroform Committee, known and practised. But the really important part of Professor Wood's paper is that in which he narrates his own experiments. The work of Professor Wood, as that of a tried and skilled experimenter, and one who has investigated the action of chloroform upon the heart more than once, must command the utmost attention. In reviewing modern physiological research on this subject, he justly says it is unanimous in averring that chloroform given diluted to the lower animals kills *qua* the respiration, *i.e.*, as Snow has well explained, by cumulation; given in concentrated vapour, it kills by provoking paralytic arrest of the heart. This result also obtains when chloroform is injected into the veins. The heart further after this arrest is found relaxed and incapable of responding to stimulation. Professor Wood states this result is so constant that he has repeatedly demonstrated it before his class. It has been the custom to destroy dogs in the laboratory by chloroform, and Professor Wood has "often noticed that death has been produced by primary cardiac arrest." Although possessed of these facts as the result of several years' work in the laboratory, it was determined, upon the publication of the Hyderabad Commission's report, to re-investigate the matter. This was done with the result that Professor Wood became more than ever impressed by the fact that chloroform can and does kill directly through primary arrest of the heart. One series of experiments went to show that the heart is *directly affected* and not reflexly, the view now commonly held by experts. A second series proved that although when large doses are given, respiration and the heart's action may cease synchronously, yet frequently the cardiac action ceases a perceptible period before the respiration comes to a standstill. The conclusions founded upon these experiments are well worthy of very careful consideration. The authors say chloroform acts as a powerful depressant poison upon both respiration and circulation; sometimes the influence is most felt at the heart, and death results from cardiac arrest; in other cases the drug paralyses primarily the respiratory centres, while in other instances it seems to act with equal force upon both medulla and heart." And, further, they are led to formulate "that cardiac arrest is specially prone to occur when chloroform is administered rapidly and in a concentrated form." It is also stated by the writers of the paper that they have each witnessed deaths from chloroform in the human subject, in which the heart ceased some while before arrest of respiration took place. They suggest that if the report of the Hyderabad Commission is not materially modified in its main contention, *viz.*, that chloroform does not cause primary cardiac arrest, the explanation may be found in some peculiarity of Indian pariah dogs, since European and American dogs unquestionably succumb to primary heart failure when allowed to inhale an unduly strong chloroform vapour.

In *The Lancet* of September 21st, 1889, we made the same suggestion as Professor Wood does now, and pointed out the possibility of the animals experimented on in Hyderabad being peculiarly resistant to the action of chloroform, and also indicated that differences in resisting power might also exist between the inhabitants of different cities. We further indicated variations in temperature as another possible cause of difference in the results of chloroform administration. There may be other factors still unknown, and which may be ascertained by further experiment. We have no doubt that the experiments of the Hyderabad Chloroform Commission and those of Drs. Wood and Hare were made with equal care, and the apparent discrepancies between the results will in the end only lead to a fuller and more perfect knowledge of the truth. But we have foreseen that it is quite impossible to come to a final conclusion regarding the action of chloroform on man from experiments, however numerous and however careful, on the lower animals. Such experiments are of great value, but the question must also be worked out from the clinical side, and it is for this reason that we have sent out a request for information regarding the results of the administration of anæsthetics. We trust that the difficulty of arriving at exact conclusions without a very large basis of facts will induce all those who can give us information to do so as fully as possible, and also to remember the proverb, "*Bis dat, qui cito dat.*" When we have received these returns we purpose to have them collated, and to devote careful consideration to the whole question of the action of anæsthetics, from its clinical as well as its experimental side.

# LETTER FROM DR. A. MITRA.

## TO THE EDITORS OF "THE LANCET."

SIRS,—The point raised by Surgeon-Major Deane in his letter published in *The Lancet* of January 25th is no doubt very important to the medical practitioner both in the United Kingdom and in other parts of Her Majesty's dominions. After the promulgation of the Hyderabad Chloroform Commission Report, the administrator of chloroform in a case which may unfortunately be fatal will be in a very awkward position before the coroner's court. We hear very often of "death from chloroform" in large hospitals in the United Kingdom. Considering that greatest care is always taken to entrust the duty to trained and experienced men, it is impossible to believe that no other factor but want of sufficient care plays part in such casualties. While fully appreciating the great and good work done by the Commission, I believe I am justified in saying that there is hardly one new recommendation contained in the practical conclusions given in the end of the Report which is not already known to the profession, and which careful surgeons do not observe in their every-day work in the operation room.

Dr. Deane says :—" For the last fourteen years I have administered chloroform precisely as the Commission recommends." I can fully bear testimony to this statement, as I had on several occasions administered chloroform when Dr. Deane was operating. I have administered chloroform in a very large number of cases. I have always held the theory that chloroform stops the respiration before it interferes with the circulation. During the administration of chloroform I never feel the pulse, but keep a careful eye on the respiration and condition of lips, &c. ; but, during the last four years, I had on two occasions to abandon the contemplated operation, and on three others to postpone it, for the danger that stared me in the face after the administration of only a few whiffs of chloroform. By the time this letter will reach you, English surgeons will no doubt have given their opinions on the question. The Hyderabad Commission deserve the highest credit, and it is not too much to say that it is one of the greatest scientific events of the nineteenth century ; but, at the same time, it must be observed that the last paragraph of its report has hardly the chance of being universally admitted, and is calculated to be positively dangerous medico-legally, as Dr. Deane has pointed out.

I am, Sirs, yours faithfully,
A. MITRA, L.R.C.P., L.R.C.S.

Kashmir, March 5th, 1890.

# REMARKS on the REPORT of the SECOND HYDERABAD CHLOROFORM COMMISSION, by JOHN G. McKENDRICK, M.D., (Professor of Physiology in the University of Glasgow); JOSEPH COATS, M.D., (Pathologist to the Western Infirmary, Glasgow); and DAVID NEWMAN, M.D., (Surgeon to the Western Infirmary, Out-door Department, Glasgow); Members of the British Medical Association Committee on Anæsthetics.

(REPRINTED FROM THE BRITISH MEDICAL JOURNAL OF JUNE 14TH, 1890.)

*The work of the Commission—The Dangers of Chloroform—Its Action on the Heart—Liability to Sudden fall of blood-pressure—Gradual Fall constantly observed—Chloroform or Asphyxia?—The Relative Importance of Heart and Respiratory Failure—Points of Agreement.*

We desire, in the first instance, to congratulate the members of the Hyderabad Chloroform Commission on the completion of an investigation of great importance to humanity. It reflects much credit not only on the members of the Commission who carried out the experiments, but also on the Government of His Highness the Nizam, his Prime Minister Sir Asman Jah, and especially on Surgeon-Major E. Lawrie, whose zeal and energy originated and successfully carried out the Commission. The completion of a scientific investigation of such magnitude, and with the aid of all the refinements of a modern physiological laboratory in the capital of the largest native Indian State, is an event of historical importance, and may be regarded as an earnest of the time when East and West will co-operate for the advancement of science.

2. The report of the Hyderabad Chloroform Commission appeared in the *Lancet* of January 18th, 1890 (vol. i., 1890, p. 149). The Reports of the British Medical Association's Committee was printed in the Journal, vol. i., 1879, pp. 1, 103, and 921; and in vol. ii., 1880, p. 957.

3. We have to thank Dr. Lawrie for kindly submitting to our inspection the kymographic tracings of blood-pressure taken by the Hyderabad Commission. These convey an idea of the enormous amount of work done by the Commission,—work involving great expenditure of time and energy, and demanding high technical skill. We delayed the present remarks till we should have the opportunity of examining these tracings, and we have made the examination with a view to adjusting our points of difference and bringing out our points of agreement.

4. The main point of difference between the Commission and the British Medical Association Committee has reference to the action of chloroform on the heart. This will be taken up afterwards; meanwhile, we may note that in our report we fully recognised the danger from failure of the respiration,—a fact which the Hyderabad Commission has omitted to allude to in referring to our report. Thus, in our report we say: "Chloroform may cause death in

dogs either by primarily paralysing the heart or the respiration." "In most cases respiration stops before the heart's action." "The danger of death from stoppage of the respiratory functions must be borne in mind in every case in which anæsthetics are given," &c. These sentences show that we recognised the danger to respiration. At the very beginning of our report we state: "Without going into detail, we may say that it soon became apparent to us that chloroform administered to dogs and rabbits has a disastrous effect on the respiratory centres; it is easy to kill one of these animals by pushing the chloroform till respiration is paralysed. In observing the rate of the heart during these experiments, it could often be determined by auscultation that its contractions were maintained after respiration had ceased. It was apparent, however, that even when failure of respiration was more directly the cause of death, the heart was, to some extent, simultaneously affected; there were even cases in which the heart appeared to fail at least as soon, if not before, the breathing."

It is evident from this that we regarded the danger to respiration as a proved and indeed a well-recognised fact; and our special object was to investigate the question of how far chloroform was dangerous to the heart. The result of our inquiries was to convince us that danger to the heart is at least an occasional occurrence, and one to be kept in mind in watching a patient under the influence of chloroform. In summing up, we put the matter thus: "The chief dangers are (1) sudden stoppage of the heart; (2) reduction of the blood-pressure; (3) alteration of the pulse respiration ratio; and (4) sudden cessation of the respiration." In this enumeration we did not intend to place the phenomena in the order of their relative frequency or importance, although we admit the sentence might bear this interpretation were it not taken in connection with the statements quoted above from the beginning of the report. The question, then, narrows itself down to this: Does chloroform ever kill by paralysing the heart? We assert that it may do so; the Hyderabad Commission hold that it never does so, and that it always kills by paralysing the respiratory centres or, in their own words, referring to experiments without recording apparatus: "In every case in which chloroform was pushed the respiration stopped before the heart."

5. Before entering on an examination of the Hyderabad observations, we may reiterate some of the points which our experiments seemed to bring out:—

(*a*) We showed that chloroform vapour has a paralysing effect on the muscular tissue of the heart and indeed on all kinds of protoplasm, when directly applied. When the heart of a frog was exposed to the direct action of chloroform vapour, it "became rapidly weaker till it ceased beating." As regards the action on the mammalian heart we make the following quotation from our report: "With a similar view, a method was devised for warm-blooded animals. Rabbits were first used and afterwards dogs. The animal was anæsthetised, then the trachea was opened, a tube introduced, and artificial respiration begun by means of a double acting pump (one cylinder forcing air in and another sucking it out). By an arrangement of India-rubber tubes, chloroform or any other anæsthetic could be introduced in the circuit between the pump and the trachea. It is to be understood that, in these experiments, the air passing into the animal's lungs was saturated with the vapour of the substance used. After artificial respiration had been set going, the heart was exposed by an incision in the middle line, which was carried by a pair of blunt scissors or boneforceps through the ensiform cartilage and lower part of the sternum. This was effected generally with no serious bleeding. It soon became apparent that, when chloroform is given in this way, there is at once a most serious effect on the heart; the right ventricle almost immediately begins to distend, the heart presently stops, with the right ventricle engorged with blood. The heart has often, in the case of rabbits, virtually come to a stand-still within a minute of the introduction of chloroform by the method described. The contrast was most striking when ether was used instead of chloroform, the other steps in the experiment being the same. Ether may be given for an indefinite period without interfering with the heart. We kept up artificial respiration with ether in the circuit for an hour, not including twenty minutes occupied in producing anæsthesia, and at the end of that time the exposed heart was beating as vigorously as at first."

It might be objected to these experiments that the dilatation of the heart, especially of its right cavities, may be due to asphyxia, but the fact that the heart is not affected in the same way by ether as by chloroform, although the other conditions of the experiment are precisely similar, meets that objection.

(*b*) During the operation of registering the blood-pressure either by the mercurial or by the spring kymograph, we observed in several instances, both in rabbits and in dogs, and especially in dogs, even when the blood-pressure was not very low, a sudden failure of the heart's action. This effect was a common occurrence during the administration of chloroform especially in the earlier stages or when the animal was recovering; it rarely happened with ethidene, and we never observed it with ether. This effect is well seen in the blood-pressure tracings from the rabbit, G and H.* During deep narcosis, these sudden variations do not occur. We were quite aware of the fact that these effects might be held to be due to a reflex mechanism, irritation of sensory nerves of the outer openings of the respiratory passages stimulating the inhibitory centre of the vagi in the medulla, and thus retarding or even arresting the action of the heart. Whatever may be the explanation, the fact remains that at certain stages in chloroform narcosis there may be sudden falls in blood-pressure due to interference with the heart's action, and no one will deny that this is a serious state of matters.

(*c*) We have seen in more than one instance the blood-pressure of the dog become enormously reduced, and the pulsations become so infrequent as to be virtually ineffectual while respiration continued. A record of this experiment produced the "Glasgow trace," as it is termed by the Hyderabad Commission.† The explanation of this trace is of great importance, and on this point we venture to differ from the Hyderabad Commission, as will appear further on.

6. The principal points in which the Hyderabad Commission differ from us are (i) the question of how death occurs under the administration of chloroform, whether the danger is only of failure of the respiration, or whether there is danger also on the side of the heart;

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* Journal, Vol. II., 1880, p. 966.

† *See* Trace A, Journal, Vol. II., 1880, p. 962, which we have here reproduced.

(ii) the explanation of the sudden effect on the heart sometimes occurring under the influence of chloroform, and observed equally by the Hyderabad Commission and by us, but ascribed by the Commission to asphyxia and by us to a peculiar, and apparently capricious, influence of chloroform.

(i) In the report of the Hyderabad Commission it is stated that chloroform "causes a gradual fall in the mean blood-pressure . . . . . As this fall continues the animal first becomes insensible, then the respiration gradually ceases, and, lastly, the heart stops." This is regarded as the normal course of events, and in confirmation of it reference is made to Experiments 168, 169, and 170. We have carefully examined these tracings, and others of a similar character. Like our own tracings, they show that, under the administration of chloroform, the blood-pressure is continually lowered, and that when chloroform is pushed the depression is apt to assume what the Hyderabad Commission admit to be a "dangerous" degree. These tracings seem to us in themselves to show that chloroform when pushed has a disastrous effect on the heart as well as on the respiration.

In most of the experiments two different kinds of tracings were taken,—one by Ludwig's and another by Fick's apparatus. The Ludwig curve shows more particularly the blood-pressure, while the Fick tracing, taken while the Ludwig tracing is suspended, shows specially the excursions produced by the individual pulsations of the heart. What we notice, specially in 169 and 170, is that there is a gradual fall of the blood-pressure, occupying four minutes in the one case and five and a-half in the other. Fick tracings, taken at intervals, show not only a reduction of pressure, but a very striking diminution in the force of the pulsations. By the time the breathing stops the pressure is reduced almost to zero, and the Fick tracings, taken immediately after the cessation of the breathing, show the merest indication of movements in the heart, whose pulsations are reduced in number to less than half. So far as the efficiency of the heart is concerned in carrying on the circulation, it has virtually ceased to act by the time the respiration has stopped, and we are inclined to believe that the inefficiency of the circulation may be, at least in part, the cause of the cessation of the respiraton; this opinion being based on the fact that the fall in blood-pressure is so extreme before the respiration stops as to interfere with the proper nutrition of the respiratory centres.

(ii) In paragraph 30, section (8) of the Hyderabad Report it is said that "the effect of involuntarily holding the breath . . . . is much more remarkable, the pressure often falling with great suddenness while the heart's action is markedly slowed." Further on, in Section (12) they say: "Complete, or almost complete, asphyxia, as by forcibly closing the nose and mouth or closing the tracheal tube after tracheotomy, has an effect similar to but more marked than that produced by holding the breath, and the character of the tracing corresponds precisely to that produced by irritation of the peripheral end of the cut vagus. The pressure falls extremely rapidly, sometimes almost to zero, and the heart's action becomes excessively slow, or even stops for a few seconds. If the Fick trace of Experiment 148 be compared with the photographic reproduction of Trace A of the Glasgow Committee it will be seen that they are identical, and that the slow action of the heart with great fall of pressure, which the Glasgow Committee attributed to some capricious action of chloroform upon the heart, was undoubtedly due to asphyxia." A number of tracings are adduced by the Hyderabad Commission in confirmation of these statements, and these we have carefully examined, with results different from those arrived at by them.

In the first place, in Experiments 103, 119, and 157, quoted in Note 10 of *Lancet* Report, we have sudden falls of pressure, and opposite some of these, not all, there is the note "holding breath". Thus, in 103 (Ludwig), between Fick 20 and 21, there is a very sudden fall, at the bottom of which is noted "holding breath". Again, Trace 119, Fick 2, shows a sudden fall, and in the Ludwig trace we have a similar note. It is the same with 157, Fick 2, (not Fick 4, as in Report of Commission). But these sudden falls of pressure are not confined to occasions when the animal holds its breath.

An examination of trace 157 is very instructive in this respect. Soon after the commencement of this tracing there is, as shown in the Ludwig trace, a sudden fall of blood-pressure, with the note, " taking off gag he suddenly held his breath, and pressure fell ". There was then a slight recovery of pressure, and trace Fick 2 was taken which starts from a low pressure, and shows a tracing somewhat like our A. Further on in the tracing comes Fick 4, which is very like that of Fick 2, so like that the Commission in their Report have misplaced the numbers, but the animal is distinctly noted as " struggling, but not holding breath ". It is noted, however, that chloroform is being given " with the cap closely applied ". If we add to this that in the cases noted the fall of blood-pressure seems to have been almost simultaneous with holding the breath, the effect on the heart can hardly be regarded as the result of asphyxia, as asphyxia is well known to induce reduction of blood-pressure only after a considerable time. (*See* further on.)

Turning to the experiments referred to in Section 12, where the breathing was artificially interrupted, we have been astonished to find that the traces do not bear out the statement of that paragraph. For example, take Experiment No. 150. The following facts are noted in regard to it. (*a*) Near the beginning of thet race we find that " holding nose and mouth " produces a distinct fall, not nearly so abrupt or great as in our A, or as in observations under " holding the breath ". (*b*) Again, at 11·55, pressure has greatly fallen under the influence of chloroform, which has rendered the animal insensitive, before the nose and mouth are stopped ; the fall continues, but not with any approach to the rapidity of those previously mentioned under " holding the breath ". (*c*) Further on, at 12·5–30, after the animal had partly recovered, and when chloroform is being given with " lots of air ", and with the animal "just insensitive, stopping the nose and mouth produces a much more gradual fall than in the previous two occasions apparently because the animal is less affected by the chloroform. (*d*) Again, at 12·9–30, the chloroform is pushed with the cap crammed over the nose, but the respiration is not obstructed, and there is a fall similar to that in *b*. (*e*) Still further on, at Fick 7, we have "mouth and nose closed ", but there is scarcely any effect on the blood-pressure and very little on the pulsation. (*f*) At Fick 10, chloroform having been stopped a while, holding mouth and nose produced little effect on the pressure, but distinct slowing of the pulse. After this the vagi are cut, and the experiment need not be followed further.

No. 151 brings out similar facts, namely, that when chloroform is pushed, whether the nose and mouth be held or not, the blood-pressure falls, and there is the additional fact that this seems to depend very little on the action of the vagi. Thus, soon after the beginning of the trace there is the note " stop nose and mouth ", which is followed by a slight fall. One minute after this the Fick tracing 3 shows great slowing of the pulse. Then the vagi are cut. Chloroform is given at 3·55, and there is the usual fall, to be followed by a rise when the chloroform is stopped. The pressure rises till 3·58, when Fick 4 is taken. After this the nose and mouth are again held, but there is little effect on the pressure for more than two minutes, after which the fall is somewhat rapid (from Fick 4 to Fick 6) till the nose and mouth are again set free. This shows the true asphyxia curve, the fall in pressure not occurring for two minutes after the respiration was obstructed.

No. 148 is mentioned as an example of asphyxia by obstructing the nose and mouth, and the tracing is described as " identical " with our A. We recognise a resemblance to our trace in the Fick tracings 2 and 6, but in these the chloroform is being pushed, and there is no obstruction to the respiration. The place where there is such obstruction is before Fick 17. At 3·54·15 we have "asphyxia by holding mouth", and there is a gradual fall for three-quarters of a minute, when Fick 17 is taken. The Fick tracing does not show the fall, and has no particular resemblance to our A, while the Ludwig trace shows that the fall here is, as in other cases of asphyxia, greatly slower than ours, taking three-quarters of a minute to accomplish a less fall than was attained in ours in about seven seconds. (Note that the drum in our observations revolved very much faster than in theirs. The time is given by a chronograph showing half-seconds in our Trace C.)

We turn now to our own tracings, from the examination of which the Hyderabad Commission infers that the sudden fall in blood-pressure is due to asphyxia. It has been indicated above that the fall in blood-pressure due to asphyxia is not sudden, but follows some time after the interruption to the respiration. This applies to our tracings ; but besides that, they show in their very form that there was here no asphyxia. In order that our statements may be followed we reproduce the Tracings A and C.

In Trace A it will be observed that the beginning of the fall is associated with what we take to be a respiratory curve, that is to say, after the fall has begun, as shown by the sudden dip, there is a gradual rise with five pulsations of the heart which is clearly identical with the respiratory curves further on in the trace, when the pressure is beginning to rise (*see* A, continued). It is to be noted that this tracing was taken at a time when the administration of chloroform had been stopped for two minutes.

If any doubt exists as to Trace A, none can exist as to Trace C. This records an observation on the same dog as Trace A, made on the same day after a partial recovery of the animal, and it forms a continuation of the same tracing. Two things will be observed in this tracing. In the first place, it is definitely noted on the sheet " Respiration continues ", although the heart is at a standstill. But, in the second place, the curve itself shows that there was no asphyxia. In the second block of C, there are the most obvious respiratory variations in the curve, and these continue right on till the rapid fall sets in. This tracing is so important that we may repeat here our original remarks on this observation :

"It is to be remembered that this animal got chloroform in the usual way, by a cloth saturated with the agent being held over his mouth and nose. He received no overdose, and the administration only lasted seventy seconds. As bearing on at least one mode of death under chloroform, the relation of the heart's action to respiration is of particular significance. The blood-pressure is enormously reduced, and the pulsations have become so infrequent as to be virtually ineffectual, yet respiration continues. But respiration stops forty seconds after the heart has resumed, the pulsations being still, however, so ineffectual that the pressure is even *minus*. We believe that the legitimate inference to be drawn is that the stoppage of respiration was not due directly to the chloroform, the inhalation of which had ceased for about forty-eight seconds. It seems likely that the failure of the heart in the first instance, and the insufficiency of its subsequent pulsations, were the cause of the failure of respiration. In such a state of the circulation, the respiratory centres would probably be insufficiently supplied with blood, and be consequently liable to cease acting. In this case, if death had occurred, it would only apparently have been due to the failure of the respiration, the primary failure being that of the heart. To what extent this may apply to human cases we do not venture to speculate."

7. We now come to a very interesting point, namely, that of determining the relative effects of a diminished activity of the heart or a diminished activity of respiration in bringing about a fatal result. In this connection, the report of the Hyderabad Commission contains many valuable suggestions. We both observed that there is still a fall of pressure after the animal had ceased to inhale chloroform. Thus, in the first recorded experiment of the British Medical Association Committee, it is noted : "Chloroform was given, and during deep anæsthesia a canula was introduced into the carotid artery, and connected with the kymograph. By the time connection with the kymograph was established, no chloroform had been given for about two minutes. On first making the connection, the pressure registered 104 millimètres, which may be regarded as nearly normal ; but now, without any fresh dose, the pressure rapidly fell to zero, with a remarkable retardation of the heart."* We quite agree with the Hyderabad Commission in the view that "this after-fall is probably due to absorption of a portion of the residue of chloroform in the air-passages after the stoppage of the inhalation." The Hyderabad Commission, however, hold that "slowing or temporary stoppage of the heart in chloroform administration is not dangerous", and that the "controlling influence of the vagus on the heart is a safeguard, and that it is the exhaustion of the nerve which is dangerous". Their theory is that when the inhibitory action of the vagus (which is known to be intermittent and not continuous) is called into play, the heart does not beat so quickly nor so strongly, that less chloroformed blood is transmitted to the respiratory centres, and that consequently, these centres are not then in danger of sudden arrest. Further, that if the vagus irritation ceases, or if the vagus becomes exhausted, the heart "bounds on again", and "the blood then becomes quickly saturated with chloroform, and an over-

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* JOURNAL, vol. II., 1880, p. 962.

dose is at once conveyed to the nerve centres". This ingenious view has much to support it, and we think that much credit is due to the Hyderabad Commission for its suggestion, as it undoubtedly explains the beneficial action of atropine as an adjunct to chloroform administration. We would remark, however, that in our opinion the Hyderabad Commission attach too much importance to it, and that it does not account for the facts in each case. For example, we observed in one case, after stoppage of respiration, while the heart continued beating, that respiration was spontaneously resumed ; and we supposed that by the cessation of respiration, less chloroform was taken into the blood, so that in a short time the respiratory centre recovered. In this case the heart was pulsating vigorously after stoppage of respiration, but as the chloroform in the air-passages was taken up and quickly eliminated and decomposed in the blood, the respiratory centres recovered. When, however, they recovered, breathing recommenced, more chloroform was introduced, and again there was danger to the respiratory centres and to the heart. Chloroform, no doubt, is taken from the pulmonary air-cells by diffusion, and this will be facilitated by inspiration, possibly by expiration ; at all events, the rapidity of absorption will not be the same when the pulmonary aircells are quiescent as when they are expanding and contracting in inspiration and expiration. Thus the movements of respiration will affect the amount of chloroform absorbed, and if, respiration becomes feeble, or even ceases, less chloroform will be taken up and time may be allowed for both the respiratory centres and the heart to recover. Thus the number and volume of the respiratory movements affect the quantity of chloroform absorbed and the number and volume of the heart beats affect the quantity of chloroform sent to the nerve centres. It is clear, therefore, that the problem is more complicated than as stated by the Hyderabad Commission, and that the physiological action will depend on so many factors as to make it extremely unlikely that the course of events will be the same in each case. Thus we have (1) the condition of greater or less asphyxia, as effected by the quantity of air mixed with the chloroform vapour ; (2) the occurrence of any inhibition of the vagus, as affecting the number and volume of the heart beats, and thus the amount of chloroform reaching the nervous centres ; the number and amplitude of the respiratory movements, as affecting the amount of chloroform absorbed into the blood ; (4) as we hold, the specific action of chloroform on the muscular structure and intrinsic nervous mechanism in the heart; and (5) the influence on the vaso-motor centre, as a factor in producing the great fall in blood-pressure. We think the Hyderabad Commission attach too much importance to one common mode of death, failure of the respiratory centres; and while we agree generally with their conclusions, which in many respects are similar to our own, we consider it unwise and unsafe in practice to pay no attention to the state of the circulation, and to observe respiration alone. We also consider it unwise to convey to the public, even through the profession, the notion that the administration of chloroform is a proceeding in which there is practically no danger. At the same time, we thankfully admit that the number of deaths following the use of so potent an agent has been remarkably small, even in the hands of those who pay little or no attention to the condition of circulation. As a matter of common prudence, and especially seeing that when respiration fails we can employ artificial means for its restoration, while if the heart fails, little or nothing can be done to avert a fatal issue, it is incumbent on every one giving chloroform to watch both the pulse and the breathing.

It may here be noted that our observations are in complete accord with those recorded by Drs. Wood and Hare, of the University of Pennsylvania, published in the *Medical News* of February 22nd, 1890, and reprinted in the *Provincial Medical Journal*, April 1st, 1890. The results of numerous experimental observations are embodied in the following paragraph :—

"The experiments which we have given show that chloroform acts as a powerful depressant poison upon both respiration and circulation, that sometimes the influence is most felt at the heart, and death results from cardiac arrest ; that in other cases the drug paralyses primarily the respiratory centres, whilst in other instances it seems to act with equal force upon both medulla and heart. So far as practical medicine is concerned, it makes little difference whether the heart stops before or just after respiration, so that those cases in which cardiac and respiratory arrest are almost simultaneous are, for the purposes of the clinician, the same as those in which heart arrest precedes respiratory paralysis. Finally, the general results of our new experiments also coincide with our previous experience in the laboratory, and with what we believe to be the general belief of physiologists—that cardiac arrest is specially prone to occur when chloroform is administered rapidly and in a concentrated form."

8. It will probably serve a useful purpose, seeing that the points on which we disagree with the Hyderabad Commission have been set forth in some detail above, if we now state in a summary form the more important matters on which we are agreed.

(I) We are agreed that death from chloroform occurs by failure of respiration, and that this is probably the most frequent mode of death.

(II) We are agreed that chloroform causes a gradual fall in the blood-pressure as registered by kymographic tracings from the carotid artery. This is the normal effect of chloroform.

(III) We are agreed that when chloroform is pushed this gradual fall may be so great as to become in itself "dangerous". This is admitted in paragraph (8) of the Hyderabad Report, where they state that after an animal has been involuntarily holding its breath "the gasping respiration which succeeds then causes very rapid inhalation of chloroform, with immediate insensibility and a rapid fall of blood-pressure, *which becomes dangerous*". (The italics are ours.)

(IV) In addition to this, which we may call the normal effect of chloroform on the heart and blood-pressure, both of us observed peculiarly sudden and unexpected falls of pressure, with slowing of the heart. We are agreed that this phenomenon occurs, and the Hyderabad tracings show that it is frequent. We differ as to the cause of its occurrence, the Hyderabad Commission ascribing it to asphyxia, while we contend that neither in the time of its occurrence after holding the breath nor in its general characters does it correspond with the fall of pressure due to asphyxia. Whatever be the explanation, the occurrence is in itself sufficiently serious, and should not be minimised as forming one of the sources of danger in the administration of chloroform.

# LEADING ARTICLE.

*The Lancet*, June 21, 1890.

In presenting to our readers this week a selection of tracings from those obtained by the Second Hyderabad Commission, we think it may be well to bring shortly again to their notice the facts about this Commission. Its object was an essentially practical one, and could not be better defined than in the words of His Highness the Nizam himself, "to save people's lives". This object Surgeon-Major Lawrie hoped to attain by showing experimentally what he, in common with his teacher Syme and many others, had found clinically, that attention to the respiration was the safeguard against death during chloroform anæsthesia. At his suggestion, the first Hyderabad Commission was appointed by the Nizam's Government, but the results were so different from those obtained by many other experimenters that we hesitated to accept them when they were forwarded to us. At Dr. Lawrie's instance a Second Commission was appointed, and the Nizam generously forwarded to us £1,000, with the request that THE LANCET would nominate an expert to assist in the investigation, and Dr. Lauder Brunton kindly acceded to our request to act as our representative. To take up the whole general action of anæsthetics, although interesting, would have occupied more time than the Commission had at its disposal, and consequently it restricted its experiments to those having a direct practical bearing.

In a subject of such vital interest as the mode of action of anæsthetics it is important that no conclusion should be accepted without the most thorough criticism, and it is with the view of enabling our readers to draw their own conclusions from the experiments of the Second Hyderabad Commission that we present them with a selection from the tracings obtained. Nearly 600 experiments in all were performed by this Commission, about 150 of them being blood-pressure, and each blood-pressure, as a rule, involving three or four tracings or more. Out of the total number of tracings, amounting to 400 or 500, we now select 32 in order to illustrate some of the most important points.

The facts on which the Commission lay most stress are that when chloroform is given to animals by inhalation, in the same way as it is given to patients during operations, the respiration invariably stops before the heart, and if the administration of the anæsthetic be stopped and artificial respiration be begun as soon as natural breathing ceases, life can invariably be restored. The great slowing of the heart's action which has been observed, more especially by the Committee of the British Medical Association, and which has been regarded as due to the action of chloroform, the Hyderabad Commission regard as due to asphyxia, inasmuch as they entirely failed to obtain this tracing by giving chloroform with free admixture of air, but were able to obtain it at will by simple obstruction of the animal's respiration. Moreover, instead of looking at this slowing of the heart's action as a cause of danger, the Commission regard it rather as a means of safety, preventing the anæsthetic from being carried too rapidly from the lungs to the nerve centres. From the observations made by the Commission there seems to be little doubt that while chloroform given with a free supply of air by inhalation does not paralyse the heart directly, yet that asphyxia with chloroform is very dangerous, and generally causes the heart to stop much more quickly than asphyxia alone would do, although to this rule there may be exceptions.

The influence of shock was very fully tested by the Commission, as will be seen from Experiments 79 and 185. It was found to be very much less than what might have been expected. These experiments are no doubt open to the objection that the operations intended to produce shock were not performed at the time when sudden death from shock is said to be most likely to occur, *viz.*, at the very beginning of the operation before any anæsthetic had been given at all, but were done after the animal had first of all been thoroughly anæsthetised and had only partially recovered from the anæsthesia. Of this objection the Commission took full cognizance, but decided not to perform operations before anæsthetics had been given, because animals are less liable to shock than man on account of their lower mental development, and the chance of obtaining evidence of shock seemed too small to justify the infliction of the pain which would necessarily have been entailed by operating without chloroform.

In THE LANCET of September 21st, 1889, we indicated that difference of temperature might have something to do with the varying results obtained at Hyderabad and elsewhere, and the Commission consequently noted the temperature of the room as well as of the animal in most of their experiments. The effect of chloroform upon animals with a fatty heart was ascertained by administering phosphorus to the animals previously, so that their organs became fatty ; but as will be seen from Experiment 79, the effect was less marked than might have been anticipated.

The lamentable case of death from the combined influence of nitrous oxide and tight-lacing which occurred in Edinburgh last autumn shortly before the Second Hyderabad Commission began their work induced them to test the effect of tight-lacing in monkeys subjected to the action of chloroform. It will be seen that death occurred rapidly. No doubt this might readily have been anticipated, and the Commission no doubt would not have tried the experiment had not the death of the unfortunate lady shortly before shown that the danger of tight-lacing when taking an anæsthetic was not sufficiently appreciated. These experiments of the Commission have caused much remark by many of our lay contemporaries, who did not seem to understand that their object had nothing to do with tight-lacing in general, but simply with tight-lacing as affecting the action of anæsthetics.

As we have already pointed out, a question of such importance is not likely to be finally settled either by clinical observation alone or experiment upon animals alone ; the two must be combined.

The Experiments of the Second Hyderabad Commission have supplied us with a mass of experimental data such as has never been obtained before, and is not likely to be obtained again, at least for many years ; but in order to have this supplemented from the clinical side, we issued with THE LANCET of March 15th a form of inquiry regarding deaths from anæsthetics which we trust that our readers will fill up and return as soon as possible, and

**that they will also aid us in our work by obtaining answers from as many trustworthy persons as they can.**

By combining the experimental and clinical data we trust that we shall be able to set finally at rest the vexed question of how death occurs during anæsthesia, and thus "to save people's lives", as the object of the Commission was expressed by the enlightened ruler to whose generosity, backed by the public spirit of his ministers, we owe the establishment of the Hyderabad Commission.

The tracings which we have reproduced were taken by connecting the carotid artery of the animal with a mercurial manometer, the float of which ascended and descended with every rise or fall in the arterial pressure, and traced upon the slowly-revolving cylinder blackened with soot the variations of the pressure upon the arterial system. As the revolutions of the cylinder were too slow to allow of the individual pulse beats being seen, a second cylinder was employed which revolved at a speed nearly ten times as great as the first; by using a wide tube, either limb of which could be shut off or opened at leisure, both manometers could either be put into communication at once with the artery, or a single one could be connected at a time. Usually only one was thus connected; the general variations of the arterial pressure are shown in the tracing taken by the Ludwig's or mercurial kymograph, termed shortly "Ludwig" in the tracings, and the pulse beats were taken by a Fick's kymograph on the quickly revolving cylinder. These tracings are indicated by the word "Fick" in the corner. In the tracings taken by Ludwig's manometer a straight line will be found here and there in place of the usual curve. These lines indicate the points where the Fick's kymograph was connected with the artery in place of Ludwig's. Each is numbered, and by reference to the corresponding tracings by Fick's kymograph the character and rate of the pulse at any of these periods can be readily ascertained.

## ARTICLE BY SURGEON-MAJOR LAWRIE.

[*From "The Lancet," June 21st*, 1890.]

WHATEVER may be the result of the labours of the Second Hyderabad Chloroform Commission, the gratitude of our profession is due to His Highness the Nizam of Hyderabad for the opportunity of scientific progress which his unbounded liberality has afforded. At the time the Commission was appointed, the *Pioneer*, the leading journal of India, wrote: "Nothing could show better the beneficent nature of the solid and friendly tie which binds together the British and the Nizam's Governments than the remarkable and cosmopolitan interest which His Highness the Nizam and his officials, one and all, have taken in bringing the question of the safety of chloroform to the proof. It redounds to the honour of the Government of India no less than to that of the administration of Sir Asman Jah, that the leading Indian State should take a principal share in the settlement of a question which concerns the welfare and happiness of the whole of mankind." Since we are in a position to show that the researches of the Commission have finally proved the safety of chloroform, it is right to ask that the Nizam and his Minister, Sir Asman Jah, together with the Minister's responsible advisor, the Nawab Mushtak Hussain, should receive public recognition as benefactors not only of the human race, but also of the animal kingdom, which, no less than mankind will derive benefit from the advantages chloroform confers in the abolition of pain and in the relief of suffering.

In considering the work of the Second Hyderabad Chloroform Commission, a very brief recapitulation is necessary. Four hundred and thirty animals were killed with chloroform by the Commission's Sub-Committee, or were subjected to experiments as to the effects of artificial respiration, with the object of confirming, elaborating, or disproving the work of the First Commission. In addition to this, 168 animals were killed by the Commission with chloroform and ether, and the effects were recorded by Drs. Lauder Brunton and Bomford with Ludwig's and Fick's manometers. The Sub-Committee's experiments confirmed the results obtained by the First Commission presided over by Dr. Hehir ; and in their turn the results of the Sub-Committee were confirmed by the experiments undertaken with the self-recording apparatus. In every case of death from chloroform the respiration stopped before the heart. The three series of experiments showed that simple chloroform poisoning causes paralysis of the respiratory centre and then gradual death, the heart being the last organ in the body to die. The exceptions to the rule that the heart retains its vitality longer than any other part of the body were found in four cases, in which more or less energetic contractions of the diaphragm occurred after the cessation of the heart's action. In one case the contractions took place after the thorax had been laid open, and when the lungs were collapsed. The Commission called these movements gasps, but solely for convenience and the want of a better term. They were no more respiratory movements than the wagging of the tail and the snapping of the jaws, which went on for ten minutes after death in two other cases, were movements of joy or of mastication. The respiratory is a coördinating centre, but there is no reason why any of the muscles should not contract spasmodically from stimuli applied to their own proper centres after the respiratory centre is paralysed. The diaphragmatic contractions in vomiting and hiccough are not generally considered respiratory movements.

With reference to the manometer experiments of the Commission, it must be borne in mind that the inhalation of chloroform necessarily causes a fall of the blood-pressure. When this was first imperfectly demonstrated by the English Commission in 1864 it was erroneously thought to be a source of danger ; and the idea was confirmed by the Glasgow Committee in 1879, which went further and attempted to prove, by means of more accurate apparatus, that not only does chloroform cause a fall of the blood-pressure, but that the fall is sometimes capricious, and that this constitutes the grave danger in chloroform administration. The Commission have shown that the capricious effects which the Glasgow Committee attributed to chloroform were produced by accidental asphyxia ; and the fact that the fall of the blood-pressure, as indicated in the Glasgow Committee's tracings, was irregular is now known to prove that there must have been irregularity of, or interference with, the respiration, in their experiments. How the fall of the blood-pressure is produced by chloroform may be left for physiologists to determine, but it must be clearly understood that it is in no sense a danger, in any case which is fit for an operation, unless it is excessive, that is to say, unless an over-dose of chloroform is inhaled.

The facts brought out by the manometer experiments of the Commission, which can be proved from the tracings, are :

1. "Chloroform, when given continuously by any means which ensures its free dilution with air, causes a gradual fall in the mean blood-pressure, provided the animal's respiration is not impeded in any way, and it continues to breathe quietly without struggling or involuntary holding of the breath. As this fall continues, the animal first becomes insensible, then the respiration gradually ceases, and, lastly, the heart stops beating (*vide* Experiment 169: Ludwig and Fick tracings). If the chloroform is less diluted the fall is more rapid, but it is always gradual so long as the other conditions are maintained ; and, however concentrated the chloroform may be, it never causes sudden death from stoppage of the heart."

2. Chloroform has no power of increasing the tendency to either shock or syncope during operations. Every operation that ingenuity could suggest, or that has ever been supposed to be dangerous under chloroform, was performed by the Commission in every stage of chloroformisation, without any effect upon the heart, the pulse, or the blood-pressure.

3. The Commission found, however, that struggling during chloroform inhalation, or anything which interfered with the breathing in any way, such as holding the breath or asphyxia, produced irregularities in the circulation and in the action of the heart (*vide* tracings of Experiment 148). Even such slight interference as is brought about by forcibly pulling the tongue forward had this effect (*vide* Tracing 185). In fact, pulling the tongue, which is constantly done when patients are supposed to be in danger from chloroform, was the only proceeding short of direct irritation of the vagus that appeared to produce shock. This would have been thought very paradoxical had it not been that previously, when Experiment 65 was reached, Dr. Bomford had proposed that the heart should be inhibited by electrical irritation of the vagus during chloroform poisoning, as he believed it would prove beneficial by postponing or preventing the fatal effects. If Tracings 65 and 117 are examined, it will be seen that inhibition of the heart's action and slowing of the circulation, with rapid fall of pressure, caused by irritation of the vagus, proved to be a safeguard, exactly as Dr. Bomford had anticipated, rather than a danger.

4. Following up the discovery regarding the vagus, the Commission found that an effect precisely similar to that caused by electrical stimulation of the vagus is produced through the same nerve : (*a*) in the holding of the breath, which occurs in the early stages of chloroform administration (*vide* Experiment 148 : Fick I. and II., readings 2, 3, 4, 6, 8, 9, and 11); (*b*) in asphyxia (*vide* Experiment 161: Fick 1, readings 14, 15, 16, and 17 ; and Experiment 156: Fick I., reading 5) ; and (*c*) sometimes after the respiratory centre is paralysed in the later stages of chloroform poisoning (*vide* Experiment 148 : Ludwig III., from 4·5 to 4·7 ; and Experiment 178, all tracings). The same effect is produced in man by identically the same causes, and is what THE LANCET of March 2nd, 1889, wrote as of " primary and

secondary syncope ". It is evidenced by pallor of the face and other symptoms, to which Mr. Battle and Mr. Hewett referred in their letters to THE LANCET of Feb. 22nd and March 1st, 1890.

5. The Commission discovered gradually (*vide* Experiment 148) that it is possible to give chloroform in such a way that full anæsthesia is produced, with a gradual fall of blood-pressure, unaccompanied by any irregularity of the heart or circulation. The Commission found that the way to do this was to ensure natural and regular respiration, without struggling, holding the breath, or asphyxia, or any interference with the breathing. It became evident in the course of the experiments that it was difficult, if not impossible, to make any animal inhale unduly strong chloroform vapour without previously making it hold its breath, asphyxiating it, or making it insensible. When an inhaler saturated with chloroform is held too close to the mouth and nose an animal holds its breath ; and in many animals, especially the rabbit or the goat, holding the breath stimulates the vagus and at once slows the heart and retards the circulation.

6. The experiments of the Commission proved that death from chloroform is invariably due to an over-dose ; and the question which next arose was, are there any circumstances which make chloroform inhalation dangerous, and, as it were, open the door to an overdose, or make the action of the drug appear capricious ?

7. It is fully demonstrated by the experiments of the Commission that struggling, holding the breath, any form of asphyxia, or any kind of interference with the breathing are dangerous in chloroform administration. Obviously either complete asphyxia or continuous holding of the breath, if this were possible, would be sure preventives of poisoning by chloroform ; but the danger in such conditions is due to the fact that they must alternate with extra vigorous and deep respiration. Struggling makes the inhalation of chloroform dangerous, because it either partially asphyxiates the patient, or alternately does this and accelerates the respiration and circulation, so increasing the amount of chloroform inhaled and hastening the rapidity with which the chloroform is conveyed to the brain and nerve-centres. Holding the breath causes asphyxia, and asphyxia produced in this or in any other manner poisons and stimulates the respiratory centre, and, on the one hand, makes it extremely obnoxious to paralysis by chloroform ; while, on the other, it leads to gasping and deep inspirations, by which an over-dose is very soon taken in. The rapidity of chloroform poisoning when asphyxia is produced is much the same as it would be if you threw a man into a pond, and held a chloroform inhaler over his head every time he came to the surface to take a breath. This is exactly what a timid or badly taught operator often does. He measures his chloroform and pours it into a patent inhaler, which he then applies to the patient's face. The patient holds his breath and becomes deadly pale, while his vagus slows the heart and circulation. Presently the patient takes a deep gasp, and as soon as the chest begins to move, the chloroformist thinks there is no danger and replaces the inhaler on his face, too probably adding another dose of chloroform as he does so. The patient's natural protective means being exhausted, he very quickly absorbs a fatal dose of chloroform, and we are subsequently told that his heart failed, that he afterwards gasped two or three times and then died. No care in measuring

the chloroform or diluting it in a mechanical inhaler is any guide whatever to the amount of the anæsthetic a patient inhales or absorbs. Moreover, breathing through an inhaler is not natural breathing.

8. The Commission has shown that the key to the safe administration of chloroform is that the breathing be natural, so as to avoid struggling and any form of respiratory embarrassment ; and that it may be so the anæsthetic must be administered in an open cone or cap, which is held far enough from the patient's face to avoid causing him to hold his breath or struggle, and into which he is at first made to blow after each inspiration, the cap being brought nearer to the face, and eventually quite close to it, as the chloroform begins to take effect and he breathes regularly.

The two tracings dated March 6th, 1890, Nos. 3 and 4, Ludwig, show very conveniently all the points claimed by the Commission. They demonstrate : (*a*) The gradual fall of blood-pressure caused by chloroform inhalation, when the chloroform is properly given, so as to entirely avoid struggling or holding the breath, &c. (*b*) The value of reflex winking when the eye is touched, as a guide to the state of anæsthesia. When this point is reached, the subject may, as a rule, be said to be ready for operation, and this, it will be seen from the tracings, occurs long before the fall of pressure, if this be regular, reaches a dangerous stage. (*c*) The effects of pushing chloroform till the stoppage of the respiration, the heart being unaffected till long after this stage is passed. (*d*) The slowing of the circulation and fall of blood-pressure caused by irritation of the vagus. No one doubts that the vagus has the same action in man as it has in animals. (*e*) The effect of electrical irritation of the vagus when the respiration has ceased ; and the slowing of the circulation, which retards the conveyance of chloroform, to the nerve centres. It is not difficult to understand how slowing of the circulation necessarily retards the conveyance of chloroform to the medulla. For example, if the pulse is sixty, and sixty atoms of chloroform are conveyed to the brain in a minute, only thirty atoms a minute will reach the brain (other things being equal) if the pulse is reduced to thirty by stimulation of the vagus. Tracings 3 and 4 of the effects of ordinary chloroform inhalation are identical up to the point where the respiration ceased. Tracing 3 then shows that the breathing having entirely ceased, stimulation of the vagus slowed the circulation from eighty-six to forty-two a minute and saved the dog's life. Tracing 4 shows how, after the stoppage of the respiration, when artificial respiration was not employed and the vagus was not irritated, the heart's action gradually ceased. Both tracings show how useless it would have been to have taken the action of the pulse as a guide. When the pulse failed, as it did at the end of Tracing 4, it was a sign of impending death. The two tracings demonstrate that by taking the respiration alone as a guide, danger could have been averted, as it always can be in the human subject under chloroform, if the breathing is carefully kept free. It is remarkable that in these two experiments the chloroform was given undesignedly or precisely the same length of time, *viz.*, 4·42 minutes. Considered by the light of the work of the Commission, these tracings prove incontestably that the slowing of the heart and circulation through the stimulation of the vagus is a safeguard in chloroform poisoning. The symptoms—pallor and loss of pulse—produced in a similar manner in man are none the less signs of the greatest danger, as they signify that the patient has not been breathing properly, or that he has been asphyxiated, or that the respiratory centre has been paralysed. They do not indicate, as has hitherto been supposed, that chloroform has any direct effect upon the heart; but whenever they occur, at whatever stage of chloroform administration, they show most unmistakably that it has been given in such a way as to interfere with the breathing.

The most important point of all in the tracings is to be found in the invariably gradual and regular fall of blood-pressure when chloroform is properly administered. This is shown in many of the Commission's experiments, especially from No. 164 to the end (*vide* 169), and in the tracings of March 6th, 1890. In tracings 3 and 4 of March 6th the fall of blood-pressure is gradual and perfectly regular, and they demonstrate the only method of chloroform administration compatible with absolute safety. Though fall of pressure is inseparable from chloroform administration, there is never the least danger if it falls regularly, and if the inhalation is stopped directly the state of the cornea shows that the patient is " under ". Regularity of the blood-pressure depends entirely upon regularity of the respiration. Any irregularity, therefore, in the fall of blood-pressure during chloroform inhalation indicate irregularity of or interference with the respiration ; and, *per contra*, any irregularity of or interference with the respiration at once causes irregularity in the fall of blood-pressure ; but as long as the respiration is regular and not interfered with, the fall of the blood-pressure will exactly correspond with it, and will cease long before a dangerous point is reached *if the inhalation is stopped when the cornea become insensitive*, or other signs show that the patient is " under ". If the respiration is kept up without struggling, holding the breath, or asphyxia, chloroform may be given slowly or quickly, freely and with perfect confidence, without the slightest risk to the patient.

From the standpoint of medical education it is a serious misfortune that ether has been allowed to displace chloroform in any of our large hospitals. The public has a right to demand that every medical man shall be able to administer an anæsthetic with safety in any part of the world ; and there is no anæsthetic which can be thus universally employed except chloroform. If ether is used as the sole recognised anæsthetic in the hospitals attached to our medical schools, students cannot learn how to give chloroform ; or if anæsthetics are only to be administered by specialists, students cannot learn anæsthesia practically ; and it is notorious that the advance of ether has been accompanied by a decrease in the employment of anæsthetics by the general practitioner, and has limited their usefulness.

In conclusion, I desire to do justice to the wisdom of my old masters by comparing the principles Syme laid down for the safe administration of chloroform,—principles which he always said he took from Sir James Simpson,—with the physiological proof the Hyderabad Commission has succeeded in determining from their experiments. In the lecture to which I have so often referred, which was published in THE LANCET of Jan. 20th, 1855, Mr. Syme states : " The points that we consider of the greatest importance in the administration of chloroform are : first, a free admixture of air with the vapour of chloroform ; secondly, we do not stint the quantity of the chloroform ; then, and this is most important, we are guided as to the effects, not by the circulation, but entirely by the respiration. We also always give chloroform in the horizontal position, taking care that there is no article of clothing constricting the neck, *and we never continue beyond the point when the patient is fully under the influence of the anæsthetic.*"

The Commission has demonstrated that the aim of the surgeon must be to give chloroform so that the blood-pressure should fall regularly throughout the whole administration, and that the blood-pressure can only be kept free from irregularities by absolute regularity of the breathing. The chloroform must therefore be inhaled in such a way that the breathing is natural and regular throughout. Feeling the pulse during chloroform inhalation is no guide whatever either to the blood-pressure or to the one thing necessary for safety, which is to keep it regular; and it has been shown above that the pulse is of no value as a sign of approaching danger, since it is only affected dangerously (*a*) when the respiration has been interfered with or (*b*) by an overdose. Lastly, in order to keep the breathing regular, the whole of the administrator's attention must be concentrated upon this point alone; and it is therefore clear that if, as is now recommended in most of the text-books, part of the chloroformist's attention is to be given to the pulse, an important element of danger comes into the administration.

We can no longer contend, with regard to chloroform, that the results of clinical experience and of experimental research do not agree. The investigations of the Hyderabad Commission have brought to light a strikingly precise and complete agreement between both. I have stated in THE LANCET of April 5th, 1890, that the late Mr. Syme's and my own form a continuous series amounting to more than 45,000 cases of almost daily (and often several times a day) chloroform administration, extending from 1847 to 1890, in which the respiration alone was taken as a guide, without one death resulting. Mr. Roger Williams has proved in THE LANCET of February 8th, 1890, from the statistics of one of the largest hospitals in London, (which, he says, may be accepted as reliable averages of all the London hospitals), in which the pulse is taken as a guide, and is carefully watched as well as the respiration that the deaths amount to one in every 1,236 administrations. We thus see that in a long series of 45,000 cases, extending over 40 years, in which the chloroformist's attention was concentrated on the respiration alone, and in which the chloroformists were students, there were no deaths at all; while in another series of 12,368 cases, in which a part of the chloroformist's attention was devoted to the pulse, and in which the chloroformists were specialists (anæsthetists), there were no less than 10 deaths,—a fraction over one in every 1,250 administrations. These clinical results correspond with the conclusions arrived at by the Hyderabad Commission, and are sufficient to show what a tremendous difference to the patient the mere method of administration may make. One of the London journals, the *St. James's Gazette*, recently published an article on the question "Is Chloroform Safe?" and answered it by saying, "It depends upon who gives it." We now know that it does indeed depend upon who gives it, but we also know that any intelligent third or fourth year's medical student may be trained to give it safely, so as to do good without the risk of evil.

I think I have shown that the Hyderabad Commission has proved Syme's principles to be true. The *rationale* of the proof and the keystone to the work of the Second Commission is to be found in the discovery of the safeguard action of the vagus nerve, and in the thorough comprehension of the significance of this fact. As soon as this was demonstrated, it became clear that chloroform and shock were not associates but incompatibles, and that the supposed capricious action of chloroform upon the heart was due either to the stimulating effect of concentrated vapour upon the nervous system, or to the effect of asphyxial blood upon the nerve centres, resulting in the exclusion of the poison from the system, and not the direct effect of the absorbed poison upon the heart or its nerves.

## APPENDIX TO ARTICLE IN *THE LANCET* OF JUNE 21st, 1890,

## BY SURGEON-MAJOR LAWRIE.

THE above paper was written before the Glasgow Committee's criticism appeared in the *British Medical Journal* of the 14th instant. I lose no time in exposing the Committee's fallacies, as I am obliged shortly to return to India.

The first point to notice in the Glasgow Committee's remarks is their reference to the paralysing effect of chloroform vapour on the muscular tissue of the heart. "Chloroform vapour has a paralysing effect upon the muscular tissue of the heart, and indeed upon all kinds of protoplasm when directly applied. When the heart of a frog is exposed to the direct action of chloroform vapour 'it became rapidly weaker till it ceased beating.'" The accuracy of this point may be conceded as far as it goes. It is only necessary to supplement it by stating that this paralysing effect is never sudden, but always gradual. "As regards the action on the mammalian heart," the Glasgow Committee performed artificial respiration by pumping air saturated with chloroform into the lungs of rabbits and dogs through an opening in the trachea. "It soon became apparent that when chloroform is given in this way there is at once a most serious effect upon the heart; *the right ventricle almost immediately begins to distend;* the heart presently stops, with the right ventricle engorged with blood." It is evident from this description that the serious effect upon the heart is not caused in the heart itself. The distension of the right ventricle must mean damage to the circulation through the lungs. It is the first effect of stasis in the lungs. Sir Joseph Lister has shown (*vide* Professor Roy's paper in the *Journal of Physiology*, 1879, vol. ii., p. 323) that when chloroform is applied directly to the web of a frog's foot stasis immediately takes place; and the same effect is undoubtedly produced in the same manner in the lungs and is sufficient to account for the ventricular engorgement which the Committee observed when they pumped air saturated with chloroform into the lungs of the rabbit. It is incorrect to imply that driving chloroform into the lungs brings it into direct contact with the heart, or that the process of pumping in the chloroformed air eliminates respiration from the inquiry.* If respiration is not to be taken into consideration in this way of giving chloroform, we should first require to have it proved that the air driven into the lungs is sufficient, or sufficiently free from poison, to keep up the respiratory function independently of the action of the chloroform. Air saturated with chloroform cannot do this. In any case, the fact stated by the Committee—that the right ventricle was first affected by distension in their experiment—is proof that it was due to something outside the heart, and not to any direct action upon the heart itself.

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[ * In Experiment 64 there are two observations of chloroform administration in which respiration as a factor is eliminated—*Vide* Fick 11 and 17.]

The Glasgow Committee ask us to believe that, when air saturated with ether was pumped into a rabbit's lungs, "*twenty minutes were occupied in producing anæsthesia*", and that it "may be given for an indefinite period without interfering with the heart". At the end of an hour's pumping "the heart was beating as vigorously as at first". It is contrary to all experience that twenty minutes should be occupied in anæsthetising a rabbit by means of air saturated with ether pumped into the lungs. The Hyderabad Commission made many experiments with ether, from among which I have selected two as examples of its effect. In the first manometer No. 106, a pariah dog weighing 25 lb., the tracing of which will be published as soon as possible, the administration of ether on a saturated sponge in a fairly close-fitting cap was commenced at 9h. 46m. 10s. on Nov. 21st, 1889. The blood-pressure rose immediately. It began to fall again at 9h. 46m. 30s., and continued to do so till death. At 9h. 47m. the cornea was insensitive. At 9h. 48m. 40s. there was rattling in the trachea. At 9h. 50m. 30s. the respiration was slow and shallow, and it stopped at 9h. 51m. At 9h. 52m. 30s. the pulse became imperceptible ; artificial respiration was kept up, and the dog's body was inverted until 9h. 57m. The thorax was then opened, and the dog was found to be dead. The artery was cut and the pressure fell to zero. The whole experiment lasted ten minutes and fifty seconds, and any one who chooses to examine the tracing can verify this description of what occurred. The second experiment took place on Nov. 26th, 1889, on two monkeys weighing 4 lbs. each, Nos. 129 and 130. In the first monkey, a measured quantity of chloroform was pumped into the lungs through an opening in the trachea by artificial respiration, much in the manner described by the Glasgow Committee. The inhalation was begun at 4h. 2m. 37s. 10cc. of chloroform were used in divided doses, and the heart stopped at 4h. 10m. 55s. The other monkey had ether in the same way. The inhalation was commenced at 4h. 2m. 24s. 15cc. of ether in divided doses were employed, and the heart stopped at 4h. 12m. 20s. In the chloroform experiment 10cc. of chloroform freely diluted with air killed a monkey in eight minutes and eighteen seconds. In the other experiment 15cc. of ether freely diluted with air killed a similar monkey in nine minutes and fifty-six seconds. The difference corresponds with the recognised difference in the strength of the two anæsthetics in the ordinary practice of surgery. With reference to the Committee's statement that the heart was beating as vigorously at the end of an hour as at first, we are not told whether there was any natural respiration when the ether administration was stopped, or even whether the animal was alive ; and in the absence of reliable information on these points we cannot consider this experiment trustworthy. Either the ether which the Committee employed had lost its strength, or else the Committee fell into the error of supposing that because the heart was still beating the ether had not interfered with its action.

The same vigorous action of the heart after prolonged anæsthesia, brought forward by the Committee in favour of ether, recently came under my notice with chloroform. On May 28th, 1890, air saturated with chloroform was administered to a rabbit in my presence, in precisely the same way as that described by the Glasgow Committee. The administration was begun at 12h. 59m. 40s. At 1h. 0m. 20s. the inhalation was stopped. There was no return of natural breathing, and the blood-pressure had fallen nearly to zero. Artificial respiration was commenced at 1h. 0m. 30s., and continued till 1h. 5m. 5s., when the artery was cut and the rabbit was thrown aside dead. The thorax was opened at 1h. 5m. 50s., and the heart was seen by several competent observers "beating vigorously and rhythmically". At 1h. 15m., it is noted, "heart still beating rhythmically; auricles and ventricles both contracting strongly, but the action of the ventricle is intermittent". At 2h. 5m., one hour after death, the auricles were rhythmically beating; the ventricles were still. This continued till 3 o'clock, two hours after death, after which no more notes were taken. In the face of these facts we cannot accept the vague statements of experiments which the Glasgow Committee first brought forward in 1879, and have repeated without confirmation in 1890.

The next part of the Glasgow Committee's paper deals with the manometer experiments of the Commission. I may observe that in all the manometer experiments of the Commission every event which is marked in the Ludwig tracings was written on the tracings, as the drum revolved, at the time it actually occurred. The Committee says :—"In the first place, in Experiments 103, 119, and 157 we have sudden falls of pressure, and opposite some of these, not all, there is the note, 'holding breath'. But these sudden falls of pressure are not confined to occasions when the animal held its breath."

This must mean, if it has any meaning at all, that some of the sudden falls of pressure in tracings 103, 119, and 157 (which will shortly be published) are not accounted for, and are due to some effect on the heart which the Commission overlooked. As a matter of fact, all the sudden falls of pressure in the three tracings were due (1) to holding the breath; (2) to struggling, which interferes with respiration; (3) to asphyxia; or (4) to electrical stimulation of the vagus. This is clearly stated on the tracings, and the Glasgow Committee ought not to have endeavoured to make a point by omitting to say so, in describing the inferences which they think may be drawn from any of these sudden falls.

The Committee proceed: "Turning to experiments referred to in section 12,* where the breathing was artificially interrupted, we have been astonished to find that the traces do not bear out the statements of that paragraph." It is impossible in this short abstract to follow the Glasgow Committee through the quotations which are put forward as "facts" to substantiate the assertion that the tracings do not bear out the statements in section 12. The complete report—with the whole of the tracings taken by the Commission—will shortly be published, and an examination of those tracings which the Glasgow Committee refer to will show that their assertion is inaccurate. One example of their inaccuracy must suffice. The Committee states "at Fick 7 (Experiment 150) we have 'mouth closed,' but there is scarcely any effect on the blood-pressure, and very little on the pulsation." It is distinctly shown on the Ludwig tracing that Fick 7 began at 12h. 17m. 12s. and ended at 12h. 17m. 40s.

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* Section 12 is as follows :—Complete, or almost complete, asphyxia, as by forcibly closing the nose and mouth, or closing the tracheal tube after tracheotomy has an effect similar to, but more marked than, that produced by holding the breath, and the character of the trace corresponds precisely to that produced by irritation of the peripheral end of the cut vagus. The pressure falls extremely rapidly, sometimes almost to zero, and the heart's action becomes excessively slow or, even stops for a few seconds. If the Fick trace of Experiment 148 be compared with the photographic reproduction of trace A of the Glasgow Committee, it will be seen that they are identical, and that the slow action of the heart with great fall of pressure, which the Glasgow Committee attributed to some capricious action of chloroform upon the heart, was undoubtedly due to asphyxia.

The mouth and nose were not closed till 12h. 17m. 35s.—just five seconds before the end of, and therefore too late to produce any marked effect in, the Fick reading. But the effect is well shown in the continuation of the observation on the Ludwig manometer. The Glasgow Committee have had every possible opportunity of examining the original tracings, and an inaccurate quotation such as that which I have called attention to is no less misleading than it is, from a scientific point of view, indefensible.

I make no comment on the comparison of the Commission's Experiment 148 with the Glasgow trace. All the tracings of Experiment 148 are published above, and anybody can form his own opinion about them, and about the Committee's observations on them.

The Committee next confuse the fall of pressure due to ordinary chloroform inhalation with the fall due to interference with the respiration. The Hyderabad Commission have stated that the fall of pressure and slowing of the pulse, from either involuntary holding of the breath or from asphyxia in chloroform inhalation, are due to vagus stimulation, reflex or direct. The Commission has given no opinion as to the cause of the fall produced by diluted chloroform alone. It is obvious, from all their experiments, that the effects of chloroform are first exerted upon the nervous tissues, the vaso-motor centre is very soon involved, the respiratory centre becomes paralysed, then the muscular tissues become affected, and last of all the heart. The fall of blood-pressure becomes dangerous if pushed to the point of paralysis of the respiratory centre. After this point is passed, if the poisoning with chloroform still continues, the fall becomes much more rapidly dangerous; the nutrition of the heart is profoundly interfered with, and the deprivation of oxygen produced by the paralysis of the respiratory centre causes it to gradually cease to act. The Glasgow Committee contend that "when chloroform is pushed, whether the mouth and nose be held or not, the blood-pressure falls, and there is the additional fact that this seems to depend very little on the action of the vagi". In support of this contention—which the Hyderabad Commission do not altogether dispute—the Committee quote from Fick readings 3 and 6 of Experiment 151. But Fick 3 of No. 151 is a reading during simple smothering, the vagi being intact. The effect of the smothering is to almost immediately lower the pressure about 25 millimètres, and to slow the pulse from 72 to 31 per minute at the same time. After the vagi are cut, when asphyxia is again produced by smothering (*vide* Fick 6), the effect is to accelerate the pulse to 105 per minute, while the pressure only falls about 5 millimètres. This difference is most assuredly due to the division of the vagi, and yet the Committee assert that the slight fall with rapid pulse in Fick 6 is "*the true asphyxia curve*", and depends "very little on the action of the vagi". In this connection, it may be well to notice what the Galasgow Committee have to say with regard to atropine. The Committee refer to the Hyderabad Commission's discovery of the safeguard action of the vagus, in chloroform poisoning, and remark that when the inhibitory action of the vagus is brought into play less chloroform is conveyed to the nerve centres; and, further, if vagus irritation ceases, or the nerve becomes exhausted, the heart "bounds on again, and the blood then becomes very quickly saturated with chloroform, and an overdose is at once conveyed to the nerve centres". The Committee continue: "This ingenious view has much to support it", and "it undoubtedly explains the beneficial action of atropine as an adjunct to chloroform administration. The action of atropine is to paralyse the vagus and produce the same dangerous condition as when the nerve is exhausted. If the Committee regard the effect of atropine as beneficial they must intend to convey that the inhibitory action of the vagus is a danger in chloroform administration when atropine is not used, *i.e.*, that the normal action of a healthy nerve is dangerous to life. The greatest living physiologists are agreed that the inhibitory action of a nerve like the vagus is not a danger to the organ inhibited, and the Hyderabad Commission goes a step further and shows that it may be a safeguard to the organism in poisoning by an anæsthetic. If the views of the Glasgow Committee are right, then a patient is in much less danger with the pulse at 105, carrying 105 atoms of chloroform to the medulla in a minute, than he is when the pulse is 31 and only 31 atoms of chloroform are being conveyed to the medulla in a minute. This is manifestly an absurdity. In actual practice the amount of atropine employed as an adjunct to chloroform administration would probably be so small as to be immaterial ; but, if it produces any effect at all, it must be to do more harm than good.

The Glasgow Committee's tracings have been reproduced in the *British Medical Journal.* They are given above for the sake of comparison with tracings 148 and others of the Hyderabad Commission. The Committee state that in the experiment which furnished Tracing C the chloroform was administered " by a cloth *saturated* with the agent being held over the mouth and nose." This exactly bears out the conclusion of the Commission. The animal got chloroform with insufficient air, and the ' Glasgow trace ', which the Hyderabad Commission produced over and over again in their experiments, was due to the stimulation of the nerve centres with asphyxial blood. It only remains to add that the Glasgow Committee's remarks on the condition of an animal whose pressure is " minus " require explanation. As they stand, it looks as if the Committee were describing the existence of pulse and respiration curves, as, indeed, they are represented in Trace C, below the basement or true zero line, the line of no pressure, which is, of course, an impossibility. The animal in Trace C was never in any danger except from the improper manner in which the chloroform was administered to it. It did not die ; and the Committee's argument about the danger of chloroform to the heart, which is based upon the faulty tracing, has its foundation, not in physiology, but solely in imagination.

In their practical conclusions the Glasgow Committee say : " We think the Hyderabad Commission attach too much importance to one common mode of death—failure of the respiratory centres ; and while we agree generally with their conclusions, which in many respects are similar to our own, we consider it unwise and unsafe in practice to pay no attention to the state of the circulation, and to observe respiration alone. We also consider it unwise to convey to the public, even through the profession, the notion that the administration of chloroform is a proceeding in which there is practically no danger." The statistics I have given elsewhere prove the great danger in chloroform administration of dividing the attention between the respiration and the circulation. For this danger the report of the Glasgow Committee in 1879 must be held to be very largely responsible. The Committee now argue as if it were wise to keep up the unfounded dread of chloroform the public have acquired, so that whenever an operation is about to be performed under chloroform the patient is nearly frightened to death beforehand, not by the operation itself, but by terror of the effect of the anæsthetic. The Committee state finally: "As a matter of common prudence, and especially seeing that when respiration fails we can employ artificial means for its restoration, while if the heart fails, little or nothing can be done to avert a fatal issue, it is incumbent on every one giving chloroform to watch both the pulse and the breathing." If this statement is to be acted upon, it is incumbent on every one giving chloroform to watch the pulse for heart failure, in spite of the fact recorded by the Committee themselves, that "if the heart fails, little or nothing can be done to avert a fatal issue." No teaching could possibly be more dangerous. Without denying that there may be conditions of the heart in which failure and death may occur in some of the processes connected with chloroform administration, just as a man with a certain condition of heart may die of heart failure from running to catch a railway train, the Hyderabad Commission has shown that sudden death from stoppage of the heart is not a risk of chloroform itself. If the surgeon is to have constantly before his mind the fear of the sudden death of his patient by stoppage of the heart from the mere action of chloroform, and is to carefully watch the pulse for signs which can only have a fatal issue, it is a physical impossibility for him to concentrate his attention on the warnings given by the respiration, by which alone danger can invariably be averted. The teaching of the Glasgow Committee is wrong, and if it is followed deaths with chloroform will be as inevitable in the future as they have been under the same circumstances in the past.

## LETTER FROM DR. R. M. WHITTINGTON LOWE, M.D.

To the Editors of "The Lancet".

Sirs,—I have read with much interest and pleasure the paper by Surgeon-Major Lawrie in your last issue, and only on one point do I venture to point out what appears to me to be an injustice to the teaching of our great master, Sir James Y. Simpson. Surgeon-Major Lawrie very properly tells us how Professor Syme used to insist on watching the respiration and taking that as the guide in the administration of chloroform; but Sir James Simpson was strong on this point as early as 1847, that is to say, very soon after he began to use chloroform. The Hyderabad Commission, generously conceived as it was, and ably carried out as it has been, has, nevertheless, done little more than to impress on the world at large what we have been taught at Edinburgh University since Simpson introduced chloroform some fifty years ago. Yet how slow has been the spread of that teaching. When did we ever see a stethoscope used in examination before the administration of chloroform? When did we ever see an inhaler, except those on Simpson's lecture table?—for which he had an affectionate regard as valuable antiques. When have we seen the chloroform measured out drachm by drachm? Does not all this go to show that we were correctly taught to judge by the *respiration* and the *effects* of the chloroform and not by any rule of thumb as to the condition of the heart and pulse or quantity given of chloroform? It has often occurred to me that supposing one had a case of sudden death while a patient was under the influence of chloroform (not necessarily the result of the anæsthetic—*see* Simpson on Anæsthesia) here in England, probably the first question of the coroner would be "How much chloroform was administered?" and how supremely ignorant and careless one would be considered if the answer given was "I have no idea", yet it would be a proper reply to a foolish question. This is a point deserving attention. As regards disease of the heart, Simpson says: "Valvular diseases are those which are most generally considered and insisted on as contra-indicating the use of chloroform. But this is really not the case, for that agent may be employed without the least danger in any valvular disease except, perhaps, disease of the mitral valve . . . There is, perhaps, no necessity for this dread after all." (Simpson's "Anæsthesia," p. 182.) No one can cherish a more grateful remembrance of the teaching and personal kindness of Professor Syme than I do, but it was Simpson who taught us, first, that chloroform should be pure; secondly, that in administering it there should be a free dilution of the vapour with atmospheric air; thirdly, not to use an inhaler; fourthly, to watch the respiration and beware of stertor; and, fifthly, not to think about the quantity but the effects of the chloroform.

I am, Sirs, yours truly,

Brighton, June 24th., 1890. R. WHITTINGTON LOWE, M.D.

## LETTER FROM SURGEON H. CHESTER NANCE.

### TO THE EDITORS OF "THE LANCET".

SIRS,—I can emphatically endorse all you say in your admirable annotation of May 3rd that "the ever-recurring paragraphs detailing death while under chloroform too often escape due notice". During an experience as house surgeon extending over nine years in this and other hospitals it has fallen to my duty to administer anæsthetics constantly. As I take the deepest interest in the subject, I hope, I may be excused for writing to you on it. It is surprising to me how any one can advocate chloroform in preference to ether. The number of deaths from chloroform recorded in *The Lancet* and *British Medical Journal* during the last three years is as follows: Sixteen in 1887, twelve in 1888, and thirteen in 1889. In the same period the same journals only record three deaths from ether, and but one from nitrous oxide. Again, in a report published in Paris, mentioned in your issue of April 12th, accidents due to chloroform are 1 in 1,236, whereas those from ether are but 1 in 12,581 cases. These facts surely speak for themselves.

In THE LANCET of Oct. 19th, 1889, under the heading "Deaths under Chloroform", you describe two deaths,—one in a child four years old. When resident medical officer at Pendlebury I administered ether to children from six years upwards, and can testify to its safety for *them*, and in the hospital here I have administered it to old men up to eighty in long cases of litholapaxy, and to men of eighty-two in strangulated hernia. I am told by the staff here that at no period of its history has a death been recorded from an anæsthetic. Ether is always used, unless contra-indicated. The chief reason why chloroform is given is because ether is far more difficult to administer than chloroform. If ether is to be given with satisfaction it requires an inhaler, and special instruction in the use of it. But ether always gives warning of danger, whereas under chloroform there may be sudden death. One point I would strongly urge is always to have a sufficient variety of anæsthetics on a table at hand, *viz.*, chloroform, ether, A.C.E., cocaine. Latterly I have added Messrs. Bartts' improved Clover's gas and ether inhaler. No one can predict with certainty which anæsthetic the patient will take best. I have had cases (generally alcoholic patients), where ether caused struggling and great excitement, but after Clover's inhaler had been changed for Ormsby's, with A.C.E., the patient quietly and quickly went under; *in such cases chloroform should never be given.* It is a pity that A.C.E. is not more generally used; for when ether fails to stimulate it is most valuable, as in ovariotomies and such like protracted cases.

In reference to* inhalers, I am sure none can compare with Clover's for ether; with it the exact percentage required can be given. Ormsby's is the best for A.C.E. mixture, Messrs. Bartts have lately made a hinged cap for me, whereby the mixture can be poured on to the sponge without removing the cap, for small quantities constantly put in are preferable to a large amount. Caution is needed in using the inhaler, for the alcohol and ether volatilise more rapidly than the denser vapour of chloroform. For chloroform administration nothing is better than a double piece of lint, whereby plenty of atmospheric air can be given. I have noticed five deaths where metal inhalers have been used. I have witnessed several deaths from chloroform, though I am thankful to say that none have occurred in my own practice, and I am convinced that they were due to an overdose and *not* due to shock from too little being given; and I would here draw attention to Dr. Hewett's admirable letter in your issue of March 1st, wherein he mentions a case of syncope arising from deep chloroform narcosis, where he went on with ether for the remainder of the operation. Surgeons now-a-days expect too much from anæsthetics. Profound anæsthesia cannot be maintained for 1½ or 2 hours without great danger occasionally, and there are many cases where it suffices to keep the patient on the verge of returning consciousness, *e.g.*, sequestrotomies and similar tedious but not delicate operations.

I would conclude by saying that ether should be always given between the ages of sixteen and sixty, except there is some good reason to the contrary, such as pulmonary, renal or arterial disease. Never, upon *any consideration whatever*, should chloroform be given for dental cases. It is always dangerous in the sitting posture for anything. I must apologise for taking up so much of your valuable space, but my excuse must be the immense importance of the subject and the alarming mortality of chloroform.

I am, Sirs, your obedient servant,

H. CHESTER NANCE,
House Surgeon.

Norwich and Norfolk Hospital,
Norwich, June 9th, 1890.

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* *Vide The Lancet*, vol. ii., p. 1220, 1888. Dr. Squire's communication on his giving ether for Liston in 1846, and his first inhaler.

## LETTER FROM Dr. Wm. HORROCKS, F.R.C.S.

To the Editors of "The Lancet."

Sirs,—The subject of anæsthetics is of such importance to surgeons that every detail in its administration must be criticised and discussed as thoroughly as possible. In your last issue Mr. Nance in a very practical letter writes, among other things, of the use of Clovers' inhaler. My own experience, after five years of surgical hospital residence, leads me to think unfavourably of that form of inhaler. In producing anæsthesia with it, the patient breathes and rebreathes his own exhaled air. Even if the air in the bag is constantly changed one frequently notices the marked cyanosis of the patient, denoting carbonic acid asphyxia as well as etherisation. This has been very forcibly impressed on me lately by a case of operation for intestinal obstruction, where the ether was given in a Clover's inhaler, with a free admixture of air. Towards the end of the operation there was marked cyanosis, and after partially recovering the patient died. It is but fair to state that the patient was sixty-three years of age, that the anæsthesia lasted rather over an hour, and that the pulse before the operation, although fair, was somewhat irregular. Allowing for all these factors, the opinion of the anæsthetist that the constructive fault of the inhaler contributed in no slight degree in causing death was concurred in by those present at the operation.

I am, Sirs, faithfully yours,  
Wm. HORROCKS, F.R.C.S.

Bradford, July 7th, 1890.

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## LETTER FROM Dr. C. RUATA.

To the Editors of "The Lancet."

Sirs,—I have read with great interest the Report of the Second Hyderabad Chloroform Commission, published in *The Lancet* of June 21st last, the more so as their results agree with those which I obtained after a long series of experiments which I performed during the years 1885-86, with the object of ascertaining the "influence of anæsthetics on the respiration." The manner of experimenting which I adopted was mainly as follows. A canula is connected by elastic tubes at one end with a bottle containing pieces of sponge saturated with the anæsthetics, at the bottom of which is inserted a tube for pumping the air from without; the other end of the canula is attached to a second canula, inserted into the trachea, and fastened to it. The main canula has two tubes springing from it, one communicating with the air and the other with a style which writes on a revolving drum. The mouth of the tube communicating with the anæsthetic has a valve which opens during inspiration and is shut during expiration; the tube communicating with the air has a valve which opens during expiration and remains shut during inspiration. This arrangement allows the air passing through the sponge charged with the anæsthetic to enter the lungs, and does not permit the air expired to enter the bottle containing the anæsthetic.

I enclose two pamphlets, which I published in 1886, in which you may see some selections from the different tracings I obtained. The conclusions I arrived at by this mode of experimenting with ether and chloroform are the following :—(1) That by administering the two anæsthetics in the manner explained, when death ensues, it is *always* caused by arrest *firstly* of the respiration, and *afterwards* of the heart. (2) That in all the animals experimented upon (rabbits, cats, and dogs) chloroform acts most powerfully on the respiration, stopping it most surely if, after the animal is completely anæsthetised, the anæsthetic is continued for some time. (3) That ether stops the respiration in frogs, rabbits, and cats, but its effects are more fugacious, so much so that the respiration is easily restored by employing the *direct* artificial respiration. (4) That ether does not stop the respiration of dogs, even after a continuous and prolonged administration of three hours. (5) That for these reasons ether is much safer than chloroform. This applies to animals; and if the same laws are applicable to man, I agree entirely with the Hyderabad Commission on the practical point that it is not the pulse that is the guide in anæsthetising, but the respiration. In regard to artificial respiration, that which I call *direct* artificial respiration consists simply in applying the mouth of the operator to the tube which communicates with the trachea, and expiring and inspiring, just as in normal respiration. This tube, as I noted before, has a lateral tube communicating with the external air, thus establishing a lateral current by which all the air contained in the lungs is pumped out by the operator's inspiration, and new pure air is pumped into the lungs with his expiration. By this means I succeeded in resuscitating all my dogs and rabbits, even though three minutes had elapsed after stoppage of the respiration ; while with any other method of artificial respiration until now employed (*indirect*), not only could I not succeed in such cases, but in the great majority of cases the *indirect* artificial respiration was unsuccessful, even directly after the stoppage of respiration. To me this kind of direct artificial respiration was the most striking feature of my experiments, and I proposed to adopt it in the case of man. This is very easily done by a metallic canula, conveniently curved at one end, like a common catheter, and provided with a lateral tube in order to establish the lateral current. The curved end, which is cut like the end of a Ferguson's speculum, is easily introduced into the trachea through the mouth and by the operator applying his mouth,—or, if objectionable, a pumping apparatus,—to the other end, natural respiration is imitated. I am quite satisfied that no other form of artificial respiration equals this inefficacy. As to the manner of its physiological action, I think that not only by this method is all the anæsthetic in the lung directly pumped out, but that the pure air pumped into it has a vivifying action on the nerve endings in the lungs, which are paralysed before paralysis of the centres of respiration sets in.

I remain, Sirs, most faithfully yours,

C. RUATA,

Prof. of Materia Medica to Perugia University.

July 2nd, 1890.

## LETTER FROM JOHN HOPE POTTER, Esq.

To the Editors of "The Lancet."

Sirs,—Surgeon-Major Lawrie, in his interesting article published in *The Lancet* of June 21st, evidently contends that the sudden fall in blood-pressure during the administration of chloroform is invariably due to a condition of asphyxia, though he admits a gradual fall in blood-pressure, unattended with danger, during the administration of this anæsthetic. I would call the attention of your readers to the fact that the condition which often ensues on the administration of chloroform is a condition of which the most apparent visible sign is pallor; asphyxia is not, however, accompanied with pallor, but with distension of systemic veins consequent on contraction of small arteries, and increase of arterial tension (blood-pressure raised), which condition again is consequent on stimulation of the vaso-motor centre due to circulation of blood deficient in oxygen. Now, this sequence of events in asphyxia produces a visible condition of lividity quite different from the pallor which is the danger signal in chloroform administration. In fact, to recapitulate, we have in asphyxia increased blood-pressure with lividity; in fatal chloroform narcosis we have the opposite condition, *viz.*, diminished blood-pressure with pallor. It appears to me the contention of Surgeon-Major Lawrie—that the danger is due to asphyxia, and not to the direct action of the chloroform vapour—is quite erroneous.

I am, Sirs, yours truly,

JOHN HOPE POTTER,

Cullompton, Devon, June 30th, 1890.

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## LETTER FROM Surgeon-Major EDWARD LAWRIE.

To the Editors of "The Lancet."

Sirs,—In *The Lancet* of July 19th, 1890, Mr. John Hope Potter states that the Hyderabad Chloroform Commission contend that "the sudden fall in blood-pressure during the administration of chloroform is invariably due to a condition of asphyxia." This is a mistake. In the paper published by me, as President of the Hyderabad Chloroform Commission, in *The Lancet* of June 21st, 1890, it is contended that the sudden falls of blood-pressure, with pallor, which are observed during the *improper* administration of chloroform, are invariably due to reflex or direct stimulation of the vagus, and that while this action of the vagus is in itself a safeguard, it always shows that the anæsthetic has been administered with insufficient air. The Hyderabad Commission has proved that asphyxia is only dangerous in chloroform administration because it makes people gasp. Mr. Potter further asserts that asphyxia raises the blood-pressure, and that this is consequent on stimulation of the vaso-motor centre. The tracings of the experiments of the Hyderabad Commission demonstrate that, contrary to the hitherto accepted belief, asphyxia lowers the blood-pressure. This is in strict accordance with what common sense would lead us to expect. It is evident that to raise the blood-pressure is to benefit the organism, and it cannot be said that asphyxia is ever beneficial. Mr. Potter is in error in supposing that asphyxia stimulates the vaso-motor centre. It is more probable that it paralyses that centre and stimulates the respiratory centre. Chloroform, on the other hand, paralyses both centres. Fortunately, it effects the respiratory centre most deeply first, so that when danger of overdosing arises warnings are given by the respiration, whereby that danger can invariably be averted.

The most important result of the labours of the Hyderabad Chloroform Commission has undoubtedly been to establish the proof that chloroform has never, under any circumstances whatever, a direct action upon the human heart ; and not the least important of the results of the Commission's work consists in the exposure of some of the sham physiology which has passed current as genuine for the last ten years. Not to speak of the condition of animals with the blood-pressure "minus," it is a well nigh irreparable misfortune that a whole generation of our profession should have been taught such physiological untruth as that the normal action of the vagus nerve is to imperil and occasionally to destroy life ; and it is degrading to reflect that one of the main uses to which this false knowledge has been applied in England since 1879 has been to throw dust in the eyes of the public, when coroners' inquests are held on the victims of chloroform administered in accordance with the erroneous principles advocated by the Glasgow Committee.

I am, Sirs, your obedient servant,

EDWARD LAWRIE, Surgeon-Major,

President, Hyderabad Chloroform Commission.

Hyderabad, Aug. 1890.

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## LETTER FROM Dr. ARTHUR H. W. HUNT, M.R.C.S., &c.

To the Editors of "The Lancet."

Sirs,—I send you a few particulars of a recent death at the Wolverhampton and Staffordshire General Hospital from the administration of ether, in which the patient died of syncope, and not from failure of the respiratory function.

A young man aged twenty was admitted under Mr. Vincent Jackson on June 19th last. He was a miller by trade, and sought relief from a swelling at the upper and outer side of his left leg. He was a stout, flabby man, and rather anæmic. The swelling turned out to be a chronic abscess. Previously to this he had always been healthy. The abscess was opened soon after admission, under chloroform, and another, which appeared a few weeks later was opened under nitrous oxide. In addition to these, two other abscesses were opened without an anæsthetic.

Last week it was found necessary to again place him under an anæsthetic, in order to scrape away some unhealthy granulations, and slit open a couple of sinuses. The house physician and assistant house surgeon were present. The house physician *pro tem.* (Mr. Bryett, M.B., Lond.) gave ether with a Clover's inhaler, and the ether was from the same bottle as that which had been successfully used for other patients. It was obtained from

Robbins, Oxford-Street, London. Deceased was not deeply under ether when I commenced and finished the operation, which only lasted two or three minutes. At no time was the ether on beyond the mark 3 on the inhaler. On telling the house physician I had finished the inhaler was put aside, and I commenced to dress the wound. After breathing normally several times deceased moaned, and then turned pale and ceased breathing. At the same time his pupils dilated. The tongue was at once drawn out with forceps, and artificial respiration begun. The chest was slapped with a wet towel, and hypodermic injections of ether and brandy administered. In addition a battery which was at hand was applied. Artificial respiration was kept up for forty minutes, but was of no avail. The post-mortem examination was made by Mr. Vincent Jackson, at the request of the coroner. There was a considerable layer of fat over the chest, and the muscles were paler than natural. Both lungs were healthy, but there were some old adhesions in the right pleural cavity. The heart was unduly covered with fat especially at its apex. The right side was dilated and full of blood, the left empty and contracted. The walls of the right ventricle were thin. The valves were healthy. The heart muscle was very pale and soft. A small piece examined under the microscope showed marked fatty changes. The jury returned a verdict accordingly.

This case is interesting, first, because it is opposed to the view that death from ether takes place from asphyxia ; and, secondly, from its sudden fatal termination, without any previous warning.

I am, Sirs, yours faithfully,

ARTHUR H. W. HUNT, M.R.C.S., &c.,
House Surgeon to the Hospital.

Wolverhampton, Sept. 2nd, 1890.

## OBSERVATIONS on the ADMINISTRATION and DANGERS of ANÆSTHETICS, by JAMES DUNLOP, M.D., Surgeon to the Royal Infirmary, Professor of Surgery at Anderson's College, and Medical Examiner in Criminal Cases, Glasgow.

In view of the recent development of this subject, circulars asking for information upon it have been issued by *The Lancet* to scientific medical men throughout the world. This circular has stimulated Dr. Dunlop to record the results of his observations on the subject made in wards 15, 23 and 24 of the Glasgow Royal Infirmary during the last ten years, and to state his opinions thereupon.

Allowing of unrecorded cases in these wards, the following opinions are based on observations of 3,500 cases within the specified period. To produce anæsthesia in this number of patients, chloroform has been principally used. In the administration, the drop method was adopted. Ether was sometimes employed ; its use by Clover's apparatus was begun on March 8th, 1882. Its use was greatly dependent on the skill and ability of the assistant, some assistants administering the drug with greater skill than others. In a general way ether was administered in the proportion of one case of ether to thirty cases of chloroform. Ether was employed preferably in cases of debility from long-standing disease of joints or from loss of blood in severe railway smashes or machinery injuries. This drug, however, was found unsuitable in operations for hernia, lithotomy, reduction of dislocations, rectal operations, and in persons suffering from pulmonary or bronchitic ailments, or addicted to habits of ntemperance.

Referring to the relative dangers of various anæsthetics at the operation or after the operation, Dr. Dunlop remarks that "chloroform is never absolutely safe, no matter how carefully or how skilfully it might be administered. Chloroform is much more dangerous than ether during operations; but after operations ether is more dangerous to life than chloroform from bronchitis or pneumonia setting in. It may be said that when pneumonia or bronchitis follows, the ether was badly administered. That may have been so. It is much more difficult to administer ether accurately than to administer chloroform accurately." On two occasions recently he encountered great difficulty with ether. In one case the breathing had almost ceased, and the pulse failed. The young woman's face had become blanched. She was dying not from asphyxia, but from weak heart action. The remainder of Dr. Dunlop's remarks are as follows:—

Every intelligent assistant I have had during the last ten years has felt happier during an operation when ether was used than when chloroform was administered. I have not seen any deaths from ether or chloroform for ten years; but I have seen many patients both in hospital and in private practice in very imminent danger to life during the administration of chloroform, the symptoms in some instances being those of impending asphyxia, in others those of defective heart action. I have laid down for my own guidance as a surgeon the rule never to have chloroform administered to a patient in my own house, even by the patient's own medical man. It is an extremely awkward and painful experience for a surgeon to have a death from chloroform in his own consulting-room.

A patient coming from the country, accompanied by the family doctor to administer chloroform in a surgeon's consulting-room, the patient to have an examination for stone, or the excision of a wen, or a reduction of dislocation at the elbow, or the breaking down of joint adhesions, is usually in a very unfavourable and unsafe condition for chloroforming. The patient is probably hot, anxious, excited, nervous, and tightly clothed, and at an early stage of the administration of the anæsthetic by the family doctor symptoms of heart danger suddenly appear, and death ensues. It is really much safer for such a patient's life to have him rest for a time in a hotel or elsewhere, and to have the anæsthetic administered there rather than in the surgeon's house. Under no circumstances, and for many reasons, should chloroform be administered by a medical man without at least a third party being present. Again, I never now administer chloroform in a dentist's room in dental cases. There is always great danger to life, and deaths have been far too numerous in the rooms of dentists. Besides nitrous oxide is so much safer in the hands of dentists than chloroform.

In the space at my disposal it is not my intention to discuss the general accuracy of what is termed Syme's views on the administration of chloroform, or to consider the conclusions arrived at by the Hyderabad Commission, which are based on experiments performed on Indian dogs and monkeys. To help *The Lancet* in its consideration of the question, how to save people's lives? I desire to contribute some evidence on the other question, why and how do some people die under the influence of chloroform?

During the last twenty years in conjunction with my colleague, Dr. Moore, as one of the medico-legal examiners for the city of Glasgow, I have had exceptionally favourable opportunities of acquiring experience of the fatal results following chloroform administration. In these years there has been a considerable number of deaths of individuals while under the influence of chloroform. The deaths which were made the subject of post-mortem examination, took place in the Royal and Western Infirmaries, in the hospital of the Barnhill Poorhouse, in the operating rooms of dentists and consulting-rooms and elsewhere of private practitioners in the city and suburbs of Glasgow, within the county of Lanark. A careful study of such details as are before me leads me to the conclusion that considering the population, its numbers and character under medical treatment, and subjected to chloroform, the proportion of fatal cases is very small, probably not more than from two and a-half to three per annum. Within the last ten years the deaths under chloroform have been more numerous than they had been in the previous decade. There have been, however, at least eight or nine deaths since January 1st, 1888, and of these seven were the subject of post-mortem examination, and in all, so far as I can learn, it was the respiration that was watched and not the pulse. The respiration gave no signs or warning of approaching danger. It was noted that the fatal results occurred in the young and middle-aged, not in children or very old men, and in females as well as males. It was further noted that some forms of surgical procedure had some peculiar influence in predisposing to a fatal result, such as operations upon joints, not, however, involving the use of the knife. Two individuals died who were having dislocations at the shoulders reduced, one a dislocation at the elbow, and three in breaking down adhesions within and about the knee-joint. In no single case was death attributable to nervous shock from the operation having been begun or completed while the patient was not fully under the influence of the anæsthetic. Some of the deaths which took place while the patients were under chloroform were not directly due to the poisonous effects of the drug itself, but to some other cause. One death was due to asphyxia from vomited matter (tripe) obstructing the air passages. The help present was neither adequate nor skilled, and the men lost their heads. Another was caused by the bursting of a small pouch (aneurysmal) in the brain, a third of an aneurysmal pouch in the first part of the aorta, both deaths taking place in the early period of the administration and while the patient was struggling. The deaths which are to be distinctly charged against chloroform are, according to the post-mortem appearances, divisible into two distinct classes. In the one and larger class the appearances are those of death by asphyxia—death through the lungs. In the other and fortunately smaller class, the appearances are those of death by syncope,—death through failure of the heart. The proportion is one death from syncope to about twelve from asphyxia. I may mention here that I think the deaths from asphyxia ought to be to a large extent preventable. The deaths from syncope are in most instances beyond the control of the highest skill and the greatest care. In order to make these observations somewhat clearer I mention the following :—

In the *deaths from asphyxia* the right side of the heart was full of liquid blood, not dark blood such as is present in asphyxia from drowning, but blood with a pinkish or magenta tinge, which seems to be characteristic of death from asphyxia by chloroform. The left side was firmly contracted and empty, the veins leading to the right side full of blood, the lungs behind engorged with liquid blood of a dark pinkish tint. A general engorgement of the veins of the liver and kidneys, and in some cases petechial spots formed by extravasated blood in the serous covering of the heart and pleural lining of the ribs, and the covering of the lungs. We find often similar petechial spots in new-born infants, who make a struggle to breathe fully and freely, and fail from obstruction by pressure, &c. In the *deaths from syncope*, or failure of heart's action, there was found a flaccid heart with all the cavities empty. No blood on the right side ; no contraction of the left ventricle. There was blood in the veins ; the cavæ were distended with blood. In some instances the heart was large, soft, and fatty, and its tissue friable. Along with this condition of heart was noted a more or less yellow, soft, fatty liver. In other instances there was no evidence of any disease of the heart or other organs ; all that was noticed was a flaccid empty heart, with venous engorgement in the lungs. In one fatal case examined by Dr. Moore there was, in addition to the empty flaccid heart, complete adhesion all round to the pericardium. Death was due to syncope from failure of the heart's action. *Deaths from asphyxia*, or preventable deaths, took place under two distinct sets of circumstances. In one set the administration of the chloroform was too long continued. The patient was too deeply under its influence from the beginning. There was an overdose, the breathing became shallower and shallower, the chest ceased to move, the diaphragm and abdominal walls only moving. The face and lips became livid, and death ensued in spite of artificial respiration. In the second or other set the patient was placed fairly under the influence of chloroform, administered with a due admixture of air, but during the operation, owing to the patient appearing to come out from under its influence, a further dose was administered, and this dose was excessive, and the patient died. The cause of death was asphyxia through the lungs. This is not an uncommon blunder, even in the hands of skilled men, and is due to want of care in the administration and the neglect of watching the respiration.

There are some conditions of lungs and other organs which render the administration of chloroform specially hazardous, and which increase the risk of death from asphyxia. One is a lung or lungs bound down to the walls of the chest and diaphragm by old adhesive bands, the patient having had extensive pleurisy. The breathing capacity or volume of the weakened lung being greatly diminished, a small or ordinary quantity becomes an overdose. With lungs in this condition a very little chloroform seems to embarrass the respiratory organs and bring about a fatal result.

In two dental cases in which chloroform was administered by experienced men, who had not, however, examined the chests, and did not know how imperfect and unsatisfactory the respiratory machinery was, death ensued early in the administration and rapidly. There was no help at hand of a proper kind to save life by artificial respiration. I have known humpbacked patients giving great trouble from imperfect breathing apparatus. Another condition which is embarrassing, but in a different way, is albumen in the urine—Bright's disease. In several of the fatal cases albumen was present in large quantity. Urine should always be examined when possible—"forewarned is forearmed." With kidney action defective in the elimination of urea, epileptiform convulsions may occur and death result. There are always danger and trouble during the administration of chloroform when albumen is present. To prevent death from asphyxia chloroform requires skill, experience, undivided attention, prompt action in time of danger, and a suitable method of administration. Anæsthetics should never be administered in a close, crowded, small, ill-ventilated apartment, or side room of a ward, or in the lobby of an operating theatre. Neither should there be a crowd around the patient witnessing the operation. Several deaths had the fatal result induced or hastened by one or other of these conditions.

Of the apparatus used in the administration of chloroform, based upon the evidence afforded by the deaths, Allis's inhaler is the most dangerous. Next in order is the folded towel arrangement, and the safest of all is the drop method on flannel stretched on wire.

Deaths from syncope or failure of heart are not common. They do, however, occur, and, as a rule, early in the administration. A small quantity of concentrated vapour without any air administered to a nervous, timid patient has proved fatal. A healthy, florid-faced young man had chloroform administered to him in private by a surgeon who had gained much experience of chloroforming in the wards of the Royal Infirmary. The chloroform was administered on a folded towel, three or four drachms having been poured on. While the patient was counting, and when he had reached the number twenty-five, his face became at once pale and blanched, the pupils suddenly widely dilated, no pulse was felt at the wrist. The young man was dead. On post-mortem examination I found his heart an empty flaccid bag. There was no disease of any organ, and no evidence of death from asphyxia. I have known a robust-looking ship captain die under precisely similar circumstances, and on post-mortem examination I found only an empty flaccid heart. Both men had hearts and nervous systems specially sensitive to chloroform, and the fatal result could not have been provided against. There is no possibility of discovering beforehand the existence of such a sensitive heart. Weakly, flabby, thin-walled, yellow hearts, and hearts bound to the pericardium by old adhesive bands of probably rheumatic origin, are prone to become paralysed very early in the administration of chloroform.

Indian dogs and monkeys, the subjects of experiments by the Hyderabad Commission, do not afford the cardiac and pulmonary complications which are often present in man, and conclusions based upon such experiments, however interesting and important they may be when viewed from a purely scientific standpoint, are of very little practical importance or value as guides to us in the safe administration of chloroform to our patients.

Strange as it may seem, disease of valves, hypertrophy of walls, cardiac murmurs of all kinds, give no trouble during the administration of chloroform. I know that from experience in the operating theatre, and I have never found any valvular disease in any case of death under chloroform. I have no post-mortem data of deaths from syncope in the last period of the administration of chloroform. On that point I have nothing to say.

In conclusion, I have to state that after thirty years' experience as a surgeon in hospital and in private practice, experience both of the living and the dead, I have formed the opinion that at all times and under all circumstances chloroform cannot be administered without some risk to the patient's life. That risk, however, may be greatly diminished—reduced to a minimum—by an accurate knowledge of the patient's condition of body and mind and the skilful administration of the anæsthetic, which includes due attention to the pulse, as well as watching the respiration.

*Glasgow.*

## LETTER FROM SURGEON H. HERBERT.

To THE EDITORS OF "THE LANCET."

SIRS,—On September 10th, while performing a lateral lithotomy on an Arab boy, about sixteen years old, it was suddenly found that the patient had stopped breathing. He had been got under perfectly quietly, and he had evidently simply got too much of the anæsthetic. It was still being administered when the stoppage of respiration occurred. Artificial respiration, with head thrown back over the edge of the table and with the ammonia bottle at the nose, was kept up for, I think, nearly two minutes. The time was not taken, however, and it may have been more or less. Then very feeble respiration began and gradually strengthened. At the beginning neither the pulse at the wrist nor heart impulse could be felt, but throughout there was a feeble flickering seen in the lines of the carotid vessels in the neck. Thus there is no doubt that respiration was mainly affected, and artificial breathing was sufficient to bring the patient round.

I am, Sirs, yours truly,

Aden, Sept. 17th, 1890. H. HERBERT, Surgeon, I.M.S.

## ANNOTATION FROM *THE LANCET.*

WE are constantly compelled to record deaths occurring under chloroform, and are generally impressed by the fact that the reports furnished are not sufficiently complete. A very natural feeling prompts reticence as regards the lay press, but when the immense importance of the matters at issue are considered, there can be no room for hesitancy about furnishing the fullest reports to the professional journals. We some months ago, in connection with the inquiry conducted by the Hyderabad Commission, issued a circular to the profession individually, and a further circular of inquiry was sent to all the hospitals and dispensaries requesting an account of personal experience of deaths under anæsthetics, and yet but comparatively few out of the great number questioned have found leisure to answer our appeal. It is admitted upon all hands that mere experimentation upon the lower animals is, without clinical experience of the behaviour of human beings under anæsthetics, inconclusive; and this is further shown by the fact that different observers arrive at different conclusions. We would, then, earnestly beg of our readers to furnish us with their own experience, and see that those responsible for the record of cases in the hospitals to which they belong duly fill in and return to us the inquiry form sent out. If this has been mislaid, we will gladly furnish fresh forms. The constant tale of deaths which are so lamentable surely claims of the profession some personal effort towards establishing a satisfactory clinical record of the behaviour of human beings under anæsthetics, and, it may be, throwing light upon the best way to avoid fatalities.

## AN ADDRESS ON ANÆSTHESIA, delivered before the International Medical Congress, Berlin, August, 1890, by H. C. WOOD, M.D., LL.D., Philadelphia.

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THE most brilliant modern achievements of the science and art of medicine in the direct saving of life are connected with surgery. These great achievements have been rendered possible by two epoch-making discoveries—antisepsis and anæsthesia. The long array of fatal cases of poisoning by carbolic acid, by iodoform, by corrosive sublimate, and by other antiseptic agents; the hundreds of deaths from chloroform, ether, and other anæsthetics, all bear witness to the verity of that strange law, in obedience to which the progress of the human race is so often at the sacrifice of the individual. Antisepsis has outgrown the dangers of its youth, and to-day the measures that are meant to save very rarely kill. On the other hand, the death roll of anæsthesia is daily added to—added to, according to my belief, at a rate that has not changed in forty years. Though this be true, from far-off Australia comes the news that jury and judge have condemned to heavy penalty a chloroformist who had lost his patient; and in England itself a well-known medical journal lends support to such a verdict by affirming that "deaths from chloroform are preventable, that with due care they may be avoided," and that, therefore, when they occur they are the result of ignorance and carelessness. If this be true, five hundred deaths and more—the result of ignorance or carelessness! Five hundred surgeons, including such names as Billroth, Jaeger, Simpson, McLeod, Agnew, Hunter McGuire, and others of equal rank, guilty of manslaughter! And still the carnage goes on. Surely, under such circumstances, the subject of anæsthesia is worthy of the attention even of this, the most learned medical gathering of the nations that the world can furnish. Antisepsis, the gift of the Old World to humanity; anæsthesia, the gift of the New World, which made the fruits of antisepsis possible—surely it is fitting that I, standing here to-day before you all as the representative of the newer civilisation, should be the chosen mouthpiece for the renewed discussion of this old but pressing theme.

In attempting a fresh study of a well-threshed-out subject, I propose to take advantage of the modern physiological methods, and to endeavour to discover by experiments upon the lower animals how anæsthetics kill, and what drugs or measures are most powerful in putting aside their lethal effects. This brings us face to face with the question. How far is it possible to adapt experiments to the needs of practical medicine, and to reason from the dog to the man? A full discussion of this subject would not be opportune, but it does seem necessary for our purpose to devote a few minutes to the pointing out of certain general guiding principles.

It ought to be acknowledged as a fundamental axiom, that no amount of experiments can overthrow a clinical fact, although when a contradiction between experimental and bedside observation seems to arise, such contradiction challenges the correctness of the alleged clinical and experimental facts alike, and should lead to careful re-examination. No amount of failure to purge a dog by elaterium proves that elaterium does not purge man ; whilst, on the other hand, the discovery that digitalis increased the blood-pressure in the lower animal very properly led to doubt as to the correctness of the, at that time general, belief that digitalis acts upon man as a cardiac sedative, and finally to the recognition of the falsity of the clinical observation upon which such belief rested.

Whatever difficulties may beset the path of the experimental therapeutist, it is certain that law is throughout the universe supreme ; that man, at least in his physical nature, is only an especially developed animal ; and if drugs act differently upon different animals, such action must be in obedience to certain laws, to us known or unknown.

Any attempt to fairly discuss these laws would lead us too far afield for the present. One law, however, treads so closely upon the matter at hand this morning, that it requires statement. This law is, that when an apparatus or system is of similar function, and of similar functional activity in different animals, the difference in the action of remedies is very rarely, if ever, in kind, though it may be in degree. Throughout mammalia the heart has one general structure and one general function ; the heart of the dog responds to the touch of digitalis precisely as does the heart of the man. The human brain is so much more highly developed than the brain of the lower mammal that it is, in fact, a new organ or apparatus, and its relation to drugs changes with the change of structure and of function. The scope of this law in regard to anæsthesia is not far to seek. The functions especially compromised in lethal anæsthesia are respiration and circulation. Surely these functions are similar throughout mammalia, and surely we ought to be able to safely reason concerning them from the dog to the man.

Recently, however, alleged clinical facts have been challenged by high authority, upon the strength of experimental results. Under these circumstances, nothing must be at once abandoned, everything must be re-examined. These re-examinations I have made, and I may be pardoned, perhaps, if I affirm that a complete study of the clinical and experimental evidence brings out, not a discord, but a most beautiful concord,—that concord between experimental and practical medicine which so often fails to appear simply because we cannot fit together the fragments of truth in our possession.

Although numerous substances have been tried, there are to-day in use practically only three anæsthetics,—nitrous oxide, ether and chloroform. Of these, nitrous oxide stands apart because it produces loss of consciousness, not by virtue of any inherent properties, but simply by shutting off from the nerve centres the supply of oxygen.

It has been asserted that the changes of circulation produced by the inhalation of nitrous oxide are essentially different from those of mechanical asphyxia, and that therefore nitrous oxide does not act as an asphyxiant. It must, however, be borne in mind that the phenomena of mechanical asphyxia are largely due to the presence of an excess of carbonic acid in the blood, whilst in the asphyxia produced by nitrous oxide there is no excess of carbonic acid, so that the phenomena present are simply the outcome of a lack of oxygen. It is, therefore, *a priori*, to be expected that the phenomena of mechanical and of nitrous oxide asphyxia should differ to a certain extent. To determine the way in which nitrous oxide inhalation affects the circulation, I have, during the past winter, in connection with my assistant and friend, Dr. David Cerna, made a long series of experiments. The result has been to show that usually the inhalation is followed by a rise of the arterial pressure, accompanied by a great disturbance of the pulse ; the pulse at first becoming irregular and tumultuous, but by-and-by settling, so that when anæsthesia is complete the pulse wave is remarkably large and full, and the rate very slow. The rise and fall of the arterial pressure in nitrous oxide anæsthesia was found to vary remarkably, not only in different inhalations, but in different periods of the same inhalation. Sometime the rise was sudden, sometimes it was slow and gradual ; sometimes it was maintained until near death, sometimes it was interrupted very early ; sometimes it was not very well marked, sometimes it was enormous.

In all our experiments respiration ceased while the heart was still in full activity. Indeed, instead of the gas acting as a cardiac depressant, it appeared to act as a cardiac stimulant, although it paralysed the vaso-motor apparatus. Thus, during complete anæsthesia, faradisation of the sciatic nerve always failed to register itself in an increase of the blood-pressure, although the heart was beating very powerfully, and although the pneumogastrics had been previously severed ; whilst late in the poisoning—at a time when the respiration had absolutely ceased, and the animal was in this respect dead, and without the power of self-recovery, and when the arterial pressure also had fallen almost to zero—the pulse waves were frequently still nearly three times the normal.

We made but few experiments as to the action of artificial respiration upon the animal dying from nitrous oxide, but these experiments proved that even after complete paralysis of the respiratory function, artificial respiration is capable of rapidly bringing the animal back to life. The heart lives on through nitrous oxide anæsthesia long after the respiratory function has been abolished, and even when the strong, full pulse fails, and the heart has almost ceased to quiver, recovery is still hopeful, because the loss of function has been caused, not by the presence of a poison, but by the absence of oxygen ; and although the paralysis may be complete, the life power sleeps before it dies, and is ready to awake at the touch of fresh oxygen.

These experimental results are in strict accord with clinical observations. The S. S. White Dental Manufacturing Company supply a very large, if not the largest, portion of the apparatus and material used for the administration of nitrous oxide in the United States ; and, in answer to my inquiry, Dr. J. W. White, their President, writes me that a computation based upon their own sales, and a knowledge of those of their rivals, has reached " the somewhat appalling result, that anæsthesia by nitrous oxide gas is probably effected in three-quarters of a million of cases annually in the United States ". Most of these inhalations have been given, not by trained physicians, but by comparatively untrained and often very ignorant dentists ; have been given to patients in a sitting or semi-sitting posture ; have been given apparently without thought or care to the general community, as the units presented themselves to the healthy and to the diseased alike ; and the result is, out of many millions of inhalations, only three deaths recorded as directly due to nitrous oxide. Could anything be safer ?

A suggestive and very practical fact which came out in our experiments is that sometimes during an inhalation of nitrous oxide the rise of the arterial pressure is extraordinary and abrupt. Not long since, in the city of Philadelphia, a gentleman arose from the dentist's chair after an inhalation of nitrous oxide, staggered, and fell in an apoplexy. Is it not easy to perceive that when the arterial system is diseased the great strain of a sudden rise of blood-pressure may produce rupture?

Some years since Dr. Kenderdine, a Philadelphia surgeon of local note, died of diabetes, which he insisted was produced in him by the inhalation of nitrous oxide. This is in accord with the researches of the French physician, Dr. Lafont, who reported a case in which sugar appeared in the urine twice in a patient after the inhalation of the gas, and who also caused in himself, and in dogs, temporary glycosuria by such inhalations. Further, Dr. Lafont noticed in a case of mitral insufficiency temporary albuminuria.

I am not aware that these very suggestive statements of the French physician have given rise to any research, except five experiments made recently upon healthy men, with negative results, by two medical students of the University of Pennsylvania, Messrs. George S. Woodward and Alfred Hand, junr. I do not believe that ordinarily the inhalation of nitrous oxide is followed by sufficient disturbance of the circulation to register itself in the urine, but the negative evidence of Messrs. Woodward and Hand is not sufficient to render it improbable that in exceptional cases the inhalation of nitrous oxide may produce albuminuria or glycosuria. Such phenomena, if they occur, are in all probability not directly produced by the nitrous oxide, but are due to the disturbances of capillary circulation caused by it.

However these facts may be, it seems to me that great caution should be used in the administration of nitrous oxide to persons, the coating of whose arteries is diseased, and it is probable that when widespread atheroma exists, ether is a safer anæsthetic than nitrous oxide.

When respiration has been suspended in nitrous oxide anæsthesia, the overwhelming indication is certainly for the employment of artificial respiration.

Notwithstanding the great safety and the many advantages which attend the anæsthetic employment of nitrous oxide, the gas can never be used for the general purposes of the surgeon, on account of the excessive fugaciousness of its influence.

The perfect anæsthetic will be a substance which has the power of paralysing the sensory-nerve trunks without affecting other functions of the body. If such drug exists it yet awaits the coming of its discoverer. Probably until such a sensory-nerve paralysant is found chloroform and ether will maintain the complete supremacy which they now have; and in the further discussion of my subject I shall confine my remarks to them. Lack of time limits this discussion to:

(1) The method in which these two drugs kill, both in man and in the lower animal, that is, whether they destroy life through the circulation or the respiration.

(2) The comparative fatality attending the use of these two agents, and the reasons for the difference.

**(3) The comparative disadvantages between the two agents, and the best method of securing the desired results.**

(4) The treatment of accidents occurring during ether or chloroform anæsthesia.

In regard to the method in which anæsthetics kill, my own teaching hitherto has been : first, that although ether in moderate doses acts as a stimulant to the circulation, yet in overwhelming amount, it is capable of depressing the heart, but that such depression of the heart is always less than the depression of the respiration, and therefore ether kills always through the respiration ; secondly, that chloroform may produce death by paralysis of the respiratory centre, or by a simultaneous arrest of respiration and circulation, but that primary paralysis of the heart may occur, and is especially prone to do so when the chloroform vapour has been given in concentrated form.

I think that these views are in accord with general professional belief, but it has recently been alleged that they are at variance with experimental evidences, so that a re-examination is necessary. What, then, are the clinical facts ?

If any credence is to be attached to the statements of competent witnesses, who have recorded human deaths during anæsthesia it is certain that in some cases, under the influence of chloroform, the pulse and respiration have ceased simultaneously ; whilst in other instances the respiration has failed before the pulse : and in still other cases the pulse has ceased its beat before the respiratory movements were arrested.

Usually ether arrests respiration in men before it paralyses the heart, but the collection of records made by Dr. J. C. Reeves certainly show that the fatal result may be produced by syncope. Thus Dr. Ernest H. Jacobs, in a report of a fatal case, asserts positively " the pulse ceased, the breathing continued ". It would seem that we must allow that ether in the human subject may cause death in the same methods as does chloroform.

Such then are the clinical facts ; or, in other words, such are the results of observations made upon the human subject. What are the results of observations made upon animals ?

The general teaching in regard to chloroform has been recently challenged by Dr. Lauder Brunton, who, as the result of 450 experiments made by himself upon the pariah dogs of India, has reached the conclusion, as published in the London *Lancet*, that, however concentrated the chloroform may be, it never causes that death from sudden stoppage of the heart. In the physiological laboratories of the University of Pennsylvania, for some years, several hundred dogs have been used annually, and a very large proportion of these dogs have been, at the end of an experiment, killed by chloroform. The observations of Dr. Reichert, professor of Physiology in the University, Dr. Hobart Hare, Demonstrator of Therapeutics, and myself, have been concordant in showing that chloroform is a cardiac paralysant, and often does kill dogs by a direct action upon the heart or its contained ganglia. The statements made concerning the Hyderabad Commission, however, led Dr. Hare and myself to a careful and thorough re-study of the subject. Some of our experiments were made by injecting chloroform into the jugular vein ; others by administering it by inhalation in the usual way.

The action of the chloroform seems to be not seriously modified by the method of administration. We definitely proved that in the dog chloroform has a distinct, direct, paralysing influence on both respiration and circulation ; that the respiration may cease before the heart beat, or the two functions be simultaneously abolished ; but that in some cases the heart is arrested before respiration. We have several times seen the respiration continues as long as one, and even two minutes after the blood-pressure has fallen to zero, and the pulse has completely disappeared from the carotid artery.

The correctness of our experiments, we claim, must be acknowledged. The experiments have not only been witnessed by a number of persons, but I have with me to-day tracings which I will gladly show any one especially interested in the subject. I do not desire to express any doubt whatever as to the correctness of the experimental data of Dr. Brunton ; I simply claim that both sets of experiments, although they have yielded different results, have been correctly and properly performed. It may be that the high heat or other climatic conditions surrounding the pariah dog make his heart less sensitive to the action of chloroform than is the heart of the dog bred in northern climates. That the thought of the different constitutions of animals in different climates is not absurd, is shown by the fact that some years ago—after I had affirmed before the Physiological Section of the International Medical Congress at London, that if certain asserted results were obtained upon European dogs, said dogs must differ from those of America, and had been met with a smile of incredulity—Dr. Brown Sequard rose and stated that he had experimented upon hundreds of dogs on both continents, and that there was a distinct difference between the animals the vascular system of the European dogs being much more developed, and operations upon them being, therefore, much more bloody than was the case with the American dog.

A very curious parallel might be traced at this point between the experimental and clinical evidence in regard to the effect of climate upon the action of chloroform. In the Southern United States chloroform is used with great freedom, and with great alleged safety ; and as long ago as 1878 Dr. Langdon B. Edwards, editor of the *Virginia Medical Monthly*, wrote : " It is one of the most peculiar effects I have ever known in medical practice—the difference of experience in Europe and the North with chloroform and ether as compared with that of the South—the high rate of mortality in the North, and the low rate in the South.". Further, in a recent letter to me Sir Joseph Fayrer affirmed the extraordinary safety of chloroform in India, and stated that he knew of no death from it as having occurred in that country, although its use is universal.

In a series of experiments which I have recently made myself to determine the changes in the circulation produced when ether anæsthesia is carried on to death, I have found that in the first periods of anæsthesia the blood-pressure is usually elevated, and that it is usually quite high at a time when the respirations are very shallow and imperfect, and the dark colour of the blood shows that it is heavily charged with carbonic acid. It is not, however, very rare for the blood-pressure to remain near the normal, and I have seen the blood-pressure begin to fall in the very first stages of ether anæsthesia ; moreover, in at least two experiments, death occurred from syncope, the respiration continuing for one or two minutes after the complete cessation of the circulation. In an experiment in which the fall of blood-pressure was most pronounced, and the arrest of the heart most complete, the dog was sick from the mange, and it is possible that the weakened heart was more susceptible than is the normal heart to the depressing influence of ether.

So far, then, as concerns the method in which ether and chloroform kill, I claim most urgently that there is no contradiction between the results as obtained by the bedside and in the physiological laboratories, and that a complete broad study of the clinical and experimental evidence leads to one conclusion, namely, that chloroform and ether are capable of paralysing the respiration and the circulation ; that in some cases one function, in other cases the other function, is primarily arrested ; but that ether is less prone to produce a primary arrest of the heart than is chloroform.

In the discussion of the second point which I have raised, namely, as to the comparative fatality attending the use of ether and chloroform, I shall not occupy time with any elaborate setting forth of the clinical evidence. In regard to the number of recorded deaths, I shall content myself with accepting the latest statistics at hand, namely, those collected by Dr. Lawrence Turnbull, who has found 375 deaths reported from chloroform, and 52 from ether. I do not believe that these figures nearly represent the total mortality ; I doubt very much whether one-third of the deaths from anæsthesia are reported ; certainly not one-third of the cases I have had personal knowledge of having been publicly recorded. Moreover, the pressure to conceal deaths from chloroform is greater than when the lethal result is due to ether. The surgeon who uses ether feels that he has employed the safest anæsthetic, and that he will receive no blame if a death occurs from it, and feels also that he has a rare case to put on record, which will give his own name permanent place in anæsthetic literature ; whereas the surgeon who uses chloroform knows that if death occur from the anæsthetic, a very large proportion of the profession, at least in the United States, will condemn him either in public or in secret for the use of this drug, and that he will be fortunate if he escape being publicly condemned by a coroner's jury. Moreover, deaths from chloroform are only too common, so that the surgeon has nothing to gain and much to lose by publication of a chloroform death, and, if possessed of the average human nature, holds his peace.

It seems to me impossible to get at the exact number of anæsthetic deaths or the proportionate fatality of ether and chloroform. Lyman considers that in regard to chloroform, the ratio of deaths to inhalations is 1 in 5,860 ; Richardson affirms that it is 1 in 2,500 to 3,000; Andrews puts it for ether at 1 in 23,204 ; and Lyman at 1 in 16,542.

Without claiming strict accuracy for any of these figures, I think that it can be asserted that the probable ratio of deaths from chloroform is three to five times that of deaths from ether.

When we come to study the effects of chloroform upon the lower animals, we find that it varies very distinctly in its action on the different species. The cat seems to withstand the fatal influences of chloroform with a power worthy of its reputed " nine lives ". Many years ago, Professor Schiff called attention to the fact that the use of chloroform as an anæsthetic in the dog is usually attended with the loss of many animals. Professor Martin, of the John Hopkin's University, writes me that the margin between complete chloroform anæsthesia in the dog, and chloroform death, is a very narrow one. This certainly is our experience in the University of Pennsylvania ; we have never been able to use chloroform as an anæsthetic without losing a very large proportion of our dogs.

Clinical and experimental results, namely, the results of experiments made in the physiological laboratory upon the lower animal, and the results of experiments made in the amphitheatre upon the higher animal (man) are again concordant. Chloroform is much more inimical than ether to animal life. The cause of this singular fatality is not, however, chiefly the cardiac action of chloroform. Chloroform is more apt to cause cardiac arrest than is ether, but it is also much more prone than is ether to cause deaths by failure of the respiration. Almost invariably, when ether is withdrawn before the dog is absolutely in the grasp of death, recovery occurs ; but over and over again I have noticed that although the chloroform was taken away whilst the respirations were still being maintained with regularity, the arterial pressure much above zero, and the pulse very apparent, yet the symptoms of cardiac and respiratory failure continued to increase until the fatal issue was reached.

It seems to me that certain general facts or principles in regard to anæsthesia must be considered as established :—

(1) That the use of any anæsthetic is attended with an appreciable risk, and that no care will prevent an occasional loss of life.

(2) That chloroform acts much more promptly and much more powerfully than ether, both upon the respiratory centres and the heart.

(3) That the action of chloroform is much more persistent and permanent than that of ether.

(4) That chloroform is capable of causing death either by primarily arresting the respiration, or by primarily stopping the heart, but that commonly both respiration and cardiac functions are abolished at or about the same time.

(5) That ether usually acts very much more powerfully upon the respiration than upon the circulation, but that occasionally and especially when the heart is feeble ether is capable of acting as a cardiac paralysant, and may produce death by cardiac arrest at a time when the respirations are fully maintained.

(6) Chloroform kills, as near as can be made out, proportionately three to five times as frequently as does ether, partly, no doubt, because it is more powerful in depressing the heart, but largely because it lets go its hold much less rapidly than does ether when inhalation ceases. Is it not possible that

> this "holding on" is because it is less volatile than ether, and can we not here get a hint why chloroform is less deadly in the South than in the North? The diffusibility of vapours or gases is in inverse proportion to the square of their densities, and the vapour of chloroform would certainly diffuse itself with far greater rapidity at 90° F. than at 70° F.

The comparative advantages and disadvantages of the two anæsthetics in practical medicine are so well known that only one or two points seem to force themselves upon our present attention. I cannot see that the surgeon is justified in putting the life of the patient to unnecessary risk of chloroformisation, except under special circumstances. I believe, moreover, that much of the unpopularity of ether is due to its improper administration. It is so easy to embarrass the respiration seriously by the folded towel, as commonly used, that not only are the struggles of mechanical asphyxia almost invariably produced, but probably death itself sometimes caused. Especially is there danger of death being thus caused mechanically in the advanced stage of etherisation, when the patient is too thoroughly etherised to struggle and when the attention of the etheriser is, it may be, attracted by some novel and difficult operation. I confess myself to once having nearly killed a patient in this way.

A proper apparatus is certainly preferable to the folded towel. Various have been invented, but, as the time is short, I shall only mention one which seems to me a practically perfect mechanism, although it is probably little known this side of the Atlantic.

The inhaler invented by Dr. O. H. Allis is based upon the theory that the patient to be etherised should be supplied with a full abundance of air, saturated with the vapour of ether. It consists essentially of a series of foldings of muslin on a wire framework, arranged almost like the gills of a fish, so as to allow the air to pass freely through, but everywhere come in contact with ether. It should be placed upon the face of the patient dry, and the ether gradually poured on from a bottle with a tight cork, pierced by two small glass tubes, one short for the entrance of the air, the other long, and reaching nearly to the bottom of the bottle for exit of ether. When properly used the Allis inhaler practically does away with the sense of suffocation, and the consequent struggles which have made etherisation alike so repulsive to patient and surgeon.

In order to determine the rapidity with which etherisation can be produced by this inhaler, Dr. M. H. Wilson kept for me notes of thirteen consecutive cases in the clinic of the Jefferson Medical College Hospital in Philadelphia. The average time required for the production of complete unconsciousness was 8 minutes. The average time during which anæsthesia was fully maintained was 32 minutes; and the average amount of ether used during this time was 7½ ounces. In twenty-one surgical cases occurring this spring in the clinical service of Professor J. William White, of the University of Pennsylvania, the average time for the production of complete anæsthesia with ether, used through Allis's inhaler was 7$\frac{9}{10}$ minutes. The results arrived at in these two clinics are so close that 8 minutes must be considered as the average time required for full etherisation by this apparatus.

In discussing the treatment of the accidents of anæsthesia, the results obtained at the bedside naturally press forward for careful consideration; but in going over the subject from this point of view, I have found so little that was novel, and so little that was satisfactory to myself, that I shall not occupy the time of this Congress with any conclusions drawn from reported cases, or personal experience in chloroform accidents. I do not think myself that the problem can be solved by any such study of cases. Death is so near and so terrible, time is so absolute, moments so important, that no surgeon would be willing or justified in waiting for the effect of any one remedy; and when a man is dosed with alcohol, nitrite of amyl, hypodermic injections of ether, digitalis, atropine, and other powerful agents; faradised, slapped, douched, stood on his head, subjected to chest movements for artificial respiration and various other measures too numerous to mention; who can tell, if by chance he recover, why he has done so? or who can point out, if by chance he die, what is the remedy whose omission or commission has led to the fatal result?

The problem is a very complex one not to be worked out amidst the excitement and responsibilities of the amphitheatre. Only in the physiological laboratory can its various elements be separated and studied each by itself, without regard to the individual life which is at stake.

In the physiological laboratory two distinct paths open, each promising to lead to some positive knowledge. We may, on one hand, enter upon the study of the minimum fatal dose of the anæsthetic, and of the results by the consentaneous or subsequent administration of its supposed physiological antagonist; or we may investigate the effect of remedies upon functions that are failing under the influences of the anæsthetic.

The objections to the first of these methods have been, in the present instance, overwhelming. The accidents seem to be independent of the amount of anæsthetic inhaled; and such a method of investigation would have required far more time than was available after I had had the honour of being asked to address this body. Death is produced by chloroform and ether through paralysis of the respiration and the heart, and the method of experimental study which I have employed consisted in a study of the action of powerful agents upon these functions when oppressed by chloroform. I have selected chloroform chiefly because it is the more powerful agent of the two anæsthetics, and the more certain in its lethal results.

The experiments have all been made upon dogs, by one plan. The carotid artery and also the trachea, having been connected with a recording drum, so that the movements of the circulation and the respiration could be consecutively recorded, the animal was anæsthetised, and when the blood-pressure had fallen almost to zero, and the respiration had ceased, or nearly ceased, as the case might be, the remedy to be tried was injected into the jugular vein, through a canula which had been previously inserted.

The more important remedies which have been used by clinicians for the averting of threatened death during anæsthesia are ether, alcohol, ammonia, nitrite of amyl, digitalis, atropine, and caffeine, alterations of position, and artificial respiration.

Although, at least in America, hypodermic injections of ether have been frequently employed even in ether accidents, such use is so absolutely absurd that it does not seem to me to require any experimental evidence of its futility. Ether in the blood acts as ether, whether it finds entrance through the lungs, through the rectum, or through the cellular tissue ; and the man who would inject ether hypodermically into a patient who is dying from ether, should, to be logical, also saturate a sponge with the ether and crowd it upon his unfortunate victim.

Instead of simply stating the results obtained in my experiments, I have thought it would be more interesting to show reproductions from some of my tracings. The first drug that I shall report upon is caffeine. I have injected it during the cardiac failure produced by chloroform, in doses varying from 3 to 7½ grains, and have never been able to perceive any distinct alteration in the arterial pressure, and no consistent distinct change of the pulse either in number or force. So far as the experiments go, they certainly indicate that the drug has no influence upon the heart that is being overpowered by chloroform. I may also state here that it is not possible in any of my tracings to make out any influence exerted by caffeine upon the respiration.

With atropine I have made a few experiments, the results being almost as negative as with caffeine. Ten cubic centimètres of a 2 per cent. solution of the atropine injected into the jugular vein of a chloroformed animal, altered the rate of the pulse beat, but had no apparent effect or influence upon the arterial pressure, or upon the respiration, and in no wise prevented final cardiac arrest.

Of all drugs, that which I think is usually most relied upon by clinicians as a cardiac stimulant in anæsthesia, as in other cases of heart failure, is alcohol. The chemical and physiological relations of alcohol to ether and chloroform are, however, so close, that many years ago I became very doubtful of the value of this drug as a stimulant to a heart depressed by an anæsthesia.

These doubts continually grew stronger from what I saw and read as to the effects of the administration of alcohol during anæsthesia, and were finally changed into conviction by the experiment of R. Dubois,* who found that in the animal to which alcohol had been freely given, much less chloroform is required than in the normal animal, to anæsthetise or to kill ; or, in other words, that alcohol intensifies the influence of chloroform and lessens the fatal dose.

In my own experiments with alcohol an 80 per cent. fluid was used, diluted with water. The amount injected into the jugular vein varied in the different experiments from 5 to 20 cubic centimètres, and in no case have I been able to detect any increase in the size of the pulse, or in the arterial pressure, produced by alcohol, when the heart was failing during advanced chloroform anæsthesia. On the other hand, on several occasions the larger amounts of alcohol apparently greatly increased the rapidity of the fall of the arterial pressure, and aided materially in extinguishing the pulse rate.

The effects of ammonia upon the failing heart of chloroform anæsthesia have been in my experiments uncertain ; sometimes distinct, although very fugacious, and sometimes imperceptible. Twenty cubic centimètres of a 10 per cent. solution of aqua ammoniæ fortior

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* *Progrès Méd.*, 1883, xi. 951.

(*United States' Pharmacopœia*), in some cases produced an immediate rise in the arterial pressure, and even fugaciously registered itself in the respiratory rate, but perhaps more frequently it failed in its influence.

The influence of injections of digitalis has been in a number of experiments very pronounced in producing a persistent gradual rise of the arterial pressure with an increase in the size of the individual pulse rate. In several instances death was apparently averted, by its injection, and I saw in one or two cases, where large amounts of the digitalis had been employed, sudden systolic cardiac arrest, indicating that digitalis, if in sufficient amount, is able to victoriously assert itself in opposition to chloroform. Moreover, when I have given chloroform to dogs whose hearts were already under the influence of digitalis, there has seemed to be a peculiar steadying or sustaining power combating the circulatory depression naturally produced by the anæsthetic, and I believe that in all cases of weak heart in man a full dose of digitalis before the administration of chloroform would greatly lessen the danger of cardiac collapse.

With the nitrite of amyl four experiments were made ; in some of these from four to ten drops of the nitrite of amyl were injected in the jugular vein ; in others the nitrite was used by inhalation. No distinct effect upon the arterial pressure was in any instance produced, and usually no alteration in the size of the pulse waves, although sometimes the pulse did appear to be a little fuller.

Of all my experimental results, those which have been reached with strychnine have been the most surprising. The injection of strychnine into the jugular vein usually produced a gradual rise of the arterial pressure, and always caused an extraordinary and rapid increase in the rate and extent of the respiration. Thus I have seen the respiration, which had practically ceased for ten seconds, suddenly, under the influence of an injection of two-tenths of a grain of strychnine, become at once very large and full, and reach a rate of 130 a minute.

A series of elaborate experiments made upon the effect of the position of the animal on the blood-pressure in the carotid and other arteries, has very clearly proved that the body of the animal whose circulation has been paralysed by chloroform acts in a measure like a tube filled with fluid. Thus, if the feet of the dog were raised vertically above the head, whilst the latter remained upon the table, an immediate rise of pressure occurred, even though the heart had ceased beating entirely ; provided that the head of the animal was kept upon a level with the table. If, however, the head of the animal was depressed below the level of the table for a distance equal to, or greater than, the length of the body of the animal, a decrease of the arterial pressure occurred at once, although the animal was in a vertical position. The phenomena observed were precisely such as would have been produced if the canula had been inserted into a tube filled with fluid, instead of the carotid artery, and the elevation and depression of this tube had registered itself on the recording drum, in obedience to the ordinary laws of hydrostatics. The phenomena were entirely independent of any beat of the heart, and were readily produced when the animal was dead, provided the death had not occurred too long previously. Sometimes, even a very few minutes after the cessation of the heart beat, it was impossible to produce the changes of pressure upon the drum. This I believe to have been due to coagulation of the blood occurring very early after death to a sufficient extent to interfere with the liquid properties of the fluid. In no case was any effect upon the respiration produced by change of position in the animal. In a number of cases, however, when the feet were elevated, the heart, which had entirely ceased beating, recommenced its work, and I have several times seen a pulse entirely disappear when the animal was taken from the vertical to the horizontal position. On the other hand, very frequently it was impossible to affect the cardiac action by changing the position of the animal. Nevertheless, the phenomena spoken of occurred too frequently to be a mere outcome of chance, though I several times noted that the heart was usually more affected by alternately elevating and depressing the feet of the animal than by keeping it in a steadily elevated or horizontal position.

When the circulation has practically ceased, under the depressing influence of an anæsthetic, inverting the body must cause the blood which has naturally collected on the enormously relaxed vessels of the abdomen, to flow into the right side of the heart and distend it, and this distension—this increase of pressure—appears at times to have a sufficient momentary influence to stimulate the failing organ.

The theory which has been advocated by some therapeutics—that inversion of the body is of value in the accidents of anæsthesia, because it causes the vital centres of the brain to be supplied with blood—is probably incorrect. The respiration in anæsthesia fails, not through want of blood in the respiratory centres, but because the blood contains a poison which paralyses these centres.

The most remarkable results which I have reached in bringing about recovery of animals to all ordinary intents and purposes dead, were obtained through the use of artificial respiration. Thus, I have seen an animal, in which no respiratory movements whatever had taken place for two minutes, and in which, during that time, no movements of blood had occurred in the carotid artery, and in which, therefore, the heart had ceased its beat, rapidly permanently restored by artificial respiration.

At one time in these researches, it appeared as though after any dose of chloroform by inhalation, the animal could be resuscitated by artificial respiration, even though heart and lungs were completely paralysed by the drug ; but finally I did find a case in which artificial respiration failed.

The results of my experiments with the lower animals may be summed up : that nitrite of amyl, caffeine, and atropine are of little or no use in chloroform poisoning ; that alcohol, when given in small amounts, has no influence, but that when given largely, materially assists in paralysing the heart and producing fatal results ; that ammonia has some little influence upon the heart, but that of all substances tried, digitalis was by far the most powerful in stimulating the failing circulation ; indeed, my experimental results indicate that it is the only known drug which is of any real practical value in such cases. Next, or perhaps even before digitalis, strychnine seems to be of value in the accidents of anæsthesia, because, whilst having some influence on the circulation, it affects powerfully the respiration. For many years chlorofrom has been used in practical medicine as the physiological and practical antagonist to strychnine, and it seems rather odd that strychnine should never have been employed as the practical antagonist to chloroform.

The one measure which in practical value far surpassed all others for the restoration of the dying animal was artificial respiration, and I have no doubt that a great majority of the deaths which have occurred in man from anæsthesia might have been avoided by the use of an active artificial respiration. The difficulty with artificial respiration as it has been hitherto practised upon man, after the Sylvester or other methods, is its inefficiency; whereas the artificial respiration which I used on animals was very active, indeed, much more efficient than natural breathing in causing circulation of air through the lungs, and therefore in removing excess of the anæsthetic from the residual air of the lungs and from the blood.

The use of what may be called "forced" artificial respiration by the physiologists, so naturally suggested a similar practice in man, that the celebrated John Hunter invented for the purpose an apparatus which consisted of a bellows so constructed that when it was extended one compartment drew in air from the lungs, while the other drew air from the atmosphere; and when it was closed the process was reversed, the fresh air being thrown into the lungs, the foul air into the atmosphere. In 1867 Richardson, of London, invented an apparatus more elegant and portable, although identical in principle with that of John Hunter's; but I have not found that either Hunter or Richardson treated by forced artificial respiration an actual case of disease or poisoning. In 1875,* Dr. John Ellis Blake reported a successful case of aconite poisoning, in which life was apparently saved, although there was no pulse for over three hours, by artificial respiration, with the use of oxygen. In this case Marshall Hall's method was at first used, but later, a small rubber tube was connected directly with a copper reservoir of condensed oxygen, the other end of the tube terminating in as mall nozzle, which was inserted in one nostril. Four hundred gallons of oxygen were thus used, but how far the force of the compressed gas was employed to dilate the lungs is not very clear; and it is doubtful whether this case should be considered as one of forced respiration. The first physician to use forced respiration in actual human poisoning, with a clear idea of its value and power, so far as my reading goes, was Dr. George E. Fell.†

It is plain that the bellows constructed by John Hunter and by Richardson are unnecessarily complex and faulty in principle. There is no need whatever of drawing the air out of the fully-filled lungs. Every physiologist knows that when the muscular system is completely paralysed by curare or even by death, the chest walls have sufficient elasticity to force air out of the lungs and all ordinary laboratory apparatus for artificial respiration is based upon this fact. For forced artificial respiration in man, an ordinary bellows of proper size is all that is required for the motive power.

The real difficulty,—the point to be especially investigated and studied,—is as to the connection between the bellows and the lungs. Hunter and Richardson simply placed a tube in one nostril, closing firmly the other nostril and the mouth of the subject.

Dr. Fell at first used a tracheal tube, the insertion of which, of course, necessitated the performance of tracheotomy. In one case, however, a simple mask covering the mouth and nostrils was a perfect success. I have had no opportunity of trying the apparatus on the living, but have made a series of experiments upon dead bodies, which have demonstrated that usually a face mask is all that is necessary for the performance of artificial respiration. Before using the mask, the tongue should be well drawn forward, and, if necessary, fixed in this position by an ordinary piece of silk suture run through it, which can be held in the hand of the operator. If in any individual case the mask fails, an intubation tube may be introduced into the larynx. I do not believe it will ever be found necessary to perform a tracheotomy.

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* *Boston Med. and Surg. Journal*, vol. xxi.

† International Medical Congress, Washington, 1887.

Dr. Fell's apparatus consists of a pair of foot-bellows by which air is forced into a receiving chamber, which is connected with an apparatus for warming the air, and a valve which can be opened and shut by a movement of the finger. This valve in turn leads to the tracheal tube. When the valve is opened the air rushes through the chamber into the lungs and expands them ; the finger is lifted, the valve shuts, the lungs contract ; and so the respiration goes on. I have no doubt that this apparatus is efficient in practice, but it is open to the serious objection of being unnecessarily complex and costly.

A much simpler, cheaper, and probably equally efficient apparatus may consist simply of a pair of bellows of proper size, a few feet of India rubber tubing, a face mask, and two sizes of intubation tubes. There should also be set in the tubing a double tube, with opening similar to that commonly found in the tracheal canula of the physiological laboratory, so that it is in the power of the operator to allow for the escape of any excess of air thrown by the bellows. This whole apparatus can be prepared at a very trifling expense, and it seems hardly necessary to point out its probable value in various narcotic poisonings, and other accidents, in which death is produced by a paralysis of the respiratory centres of temporary nature. The proper use of it,—at least with the face mask,—could be taught to persons without special medical skill, so that it not only ought to form a part of the surgeon's outfit, but might be of great service in life-saving stations, about gas works, &c.

In conclusion, I may be allowed to state that if the results and deductions arrived at in this address are, as I believe, correct, the rules for the proper treatment of accidents during anæsthesia can be summed up in a very few words :

Avoid the use of all drugs, except strychnine, digitalis and ammonia.

Give the tincture of digitalis hypodermically.

Draw out the tongue, and raise up the angle of the jaw, and see that the respiration is not mechanically impeded.

Invert the patient briefly and temporarily.

Use forced artificial respiration promptly, and in protracted cases employ external warmth and stimulation of the surface by the dry electric brush, &c., and, above all, remember that some at least, and probably many, of the deaths which have been set down as due to chloroform and ether have been produced by the alcohol which has been given for the relief of the patient.

# CLINICAL LECTURE BY SURGEON-MAJOR E. LAWRIE,

Gentlemen,—In the case of operation for the removal of the uterus and its appendages for sarcoma, which you witnessed yesterday, chloroform was administered, and the patient was kept fully anæsthetised for one hour and a-half by Miss R. Furdonji, a fifth-year Parsi lady student, without assistance or interference from anybody. You are aware that, while I hold myself entirely responsible for the safety of my patients, chloroform is always given here by students with complete immunity from accidents, and you may find it difficult to understand why there should be any difference of opinion as to its safety. Unfortunately, a difference of opinion does exist, and it is my duty to indicate to you the fallacies contained in the latest addition to the controversy on anæsthetics,—the address on anæsthesia at the Berlin International Congress. In the opening paragraph of his address Professor Wood says : "The death-roll of anæsthesia is daily added to—added to, according to my belief, at a rate that has not changed in forty years. Though this be true, from far-off Australia comes the news that judge and jury have condemned to heavy penalty a chloroformist who had lost his patient" and in England itself a well-known medical journal lends support to such a verdict by affirming that death from chloroform are preventable, that with due care they may be avoided, and that, therefore, when they occur they are the result of ignorance and carelessness. If this be true, five hundred deaths and more,—the result of ignorance and carelessness ! Five hundred surgeons, including such names as Billroth, Jaeger, Simpson, McLeod, Agnew, Hunter McGuire, and others of equal rank, guilty of manslaughter ! And still the carnage goes on." Professor Wood scouts the idea that all the deaths that have occurred from chloroform are due to ignorance and carelessness, and in a strictly limited sense he is right. The majority of the deaths from chloroform have been due to wrong teaching, and it is not the unfortunate chloroformist in far-off Australia who ought to be punished for losing his patient, but those leaders of our profession who, by their obstinate and persistent advocacy of erroneous principles, send forth from our medical schools year after year numbers of young medical men who are ignorant of the proper method of chloroform administration, and are confessedly unable to administer it with safety. A portion of Professor Wood's address consists of an attempt to divert the question of anæsthesia into a side issue on the qualities of different races of dogs and he puts forward the amazing and ridiculous contention that the heart of a dog in India is much less sensitive to chloroform than the heart of a dog bred in northern climates. If this were true, it would only be necessary to raise the temperature of our operating rooms to 80° to ensure perfect safety. Operations must in future be performed in the hothouse, that is all. Professor Wood says : "Further, in a recent letter to me, Sir Joseph Fayrer affirmed the extraordinary safety of chloroform in India, and stated that he knew of no death from it as having occurred in that country, although its use is universal." Sir Joseph Fayrer left India in 1871. Since Sir Joseph left that country the old Scotch principle of chloroform administration which was in his time almost universal has been largely replaced by other principles, and several deaths have taken place. I know of six, and five of the six have occurred since 1879.

Sir Joseph Fayrer's letter therefore merely confirms my opinion that the report of the Glasgow Committee, which was published in 1879, and was founded upon insufficient experiments, has done infinite harm, and is chiefly responsible for the fatality from chloroform during the last ten years. Dr. James Dunlop also states :* "Within the last ten years the deaths under chloroform in Glasgow have been more numerous than they had been in the previous decade."

Professor Wood next proceeds to re-examine the clinical facts. "If any credence is to be attached to the statements of competent witnesses who have recorded human deaths during anæsthesia, it is certain that in some cases under the influence of chloroform the pulse and the respiration have ceased simultaneously, whilst in other instances the respiration has failed before the pulse, and in still other cases the pulse has ceased its beat before the respiratory movements were arrested." Nothing is more certain, however, than that it is unsafe to attach credence to those whom Professor Wood styles the competent witnesses who have recorded human deaths during anæsthesia. The reports of chloroform deaths are utterly worthless and unreliable, because there is no independent person present who can take notes, and who will not interfere, although he sees the inevitable death approaching. Professor Wood then compares his clinical facts, which you now see are of no value, with the results of observations made on animals. He states that he and his colleagues definitely proved that in "the dog chloroform has a distinct, direct paralysing influence on both respiration and circulation : that the respiration may cease before the heart beat, or the two functions be simultaneously abolished ; but that in some cases the heart is arrested before respiration. We have several times seen the respiration continue as long as one and even two minutes after the blood-pressure has fallen to zero, and the pulse has completely disappeared from the carotid artery."

Professor Wood here makes the same mistake as was made by Dr. Lauder Brunton on the very last day of the Hyderabad Commission's experiments. No one who was present will forget the exciting scene when Dr. Brunton, who was watching the Fick manometer, called out that the heart had stopped, though the dog was still breathing. The excitement reached a climax when a needle thrust into the heart showed that it was beating vigorously ; and we restored the animal easily. On another occasion everybody in the room thought a dog was dead when it was not. The fact that there is no carotid pulse is no proof that the heart has stopped. Professor Wood continues : "The correctness of our experiments we claim must be acknowledged. I do not desire to express any doubt whatever as to the correctness of the experimental data of Dr. Brunton. [Why not of the Hyderabad Commission ?] I simply claim that both sets of experiments, although they have yielded different results, have been correctly and properly performed." The results being irreconcilable, either the experimental data of Professors Wood and Hare or those of the Hyderabad Commission must be incorrect. The question we have to decide is which of the two are wrong. Professor Wood asserts that his experimental data correspond with his clinical facts. But his clinical facts prove nothing whatever, except that, if part of the chloroformist's attention is devoted to the pulse in chloroform administration, deaths occur. Professor Wood's experimental data, like his clinical facts, do not prove that chloroform directly affects the heart.

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* *Vide* THE LANCET of Sept. 27th, 1890.

The cases he brings forward as instances of death from stoppage of the heart in animals are not supported by any evidence that the heart had stopped beating when he assumed that it had because the blood-pressure was low and the carotid pulse could not be felt. We must have more information regarding Professor Wood's methods before we can accept his conclusions. He probably used a mercurial manometer. In our experiments the pulse was often visible on the Fick, or glycerine, manometer long after it had disappeared on the mercurial manometer ; and the needle in the heart would beat long after the Fick tracing was reduced to a straight line.

The clinical facts which led to the formation of the Hyderabad Chloroform Commission are very different from those of Professor Wood. They consist of an almost unbroken series of 45,000 cases of chloroform administration, extending over forty years. In this long series of cases the chloroformists were guided as to the effect of the chloroform entirely by the respiration, and there was not a single death. In strict accordance with these clinical facts the experimental data of the Hyderabad Commission prove (1) that the administration of chloroform is free from risk if the breathing is perfectly regular throughout and the inhalation is stopped as soon as the animal is fully under its influence ; (2) that chloroform never causes death by sudden stoppage of the heart ; (3) that death from chloroform is always the result of an overdose ; (4) that the danger of overdosing is enormously increased by holding the breath, struggling, asphyxia, or anything which causes the patient or animal to take gasping inspirations ; and (5) that the inhibitory action of the vagus nerve, which is called into play in threatened and actual poisoning with chloroform, is a safeguard. The safeguard action of the vagus was discovered by Dr. Bomford, and its true meaning was subsequently worked out by Drs. Bomford and Brunton, and no physiological discovery that has been made of late years can compare with it in practical importance. It was never mentioned or alluded to at the Berlin Congress.

We are now able to institute a comparison between the value of Professor Wood's clinical and experimental data and that of the clinical and experimental data of the Hyderabad Commission. Professor Wood's data lead to nothing more than an admittedly unsafe method of chloroform administration. The Hyderabad Commission demonstrates that by proper attention to the breathing chloroform can be administered with safety in any part of the world. No one is in a better position than I am to appreciate the worth and significance of the Hyderabad Commission's researches. For twenty years before the Commission was appointed I had given chloroform and taught hundreds of students to give it with unvarying safety. I took no part in Drs. Brunton's and Bomford's experiments, but I had the supreme satisfaction of looking on and watching the gradual development of the proof of the truth of Syme's principles. I now know scientifically, what I knew before empirically and it is difficult to over-estimate the difference. Formerly I taught my students to be on the alert for the warnings which are given by the respiration in chloroform administration, by which danger can be averted. We frequently gave patients overdoses, and were obliged to draw forward their tongues, or occasionally even to employ artificial respiration to restore them. I now teach you that the true art of giving chloroform,—an art which any intelligent medical man can acquire,—consists not only in concentrating your attention on the breathing, but in keeping it absolutely regular throughout the administration, and in stopping the inhalation whenever the breathing is irregular, and directly the patient is fully under the influence of the anæsthetic. We never have trouble now from overdosing; you rarely see respiratory embarrassment in my operations, and during the last year, since the Commission completed its work, we have not had a case in which stoppage of the respiration has occurred. Professor Wood finds ether is as bad as chloroform in the laboratory. Clinically it does not kill so many people, because it is not pushed so far. In many cases, partial anæsthesia is considered sufficient when ether is used. Professor Wood has found out also that atropine, caffeine, and alcohol are not beneficial in chloroform administration, and that digitalis is. He has only to study our report and the vagus experiments to understand why. Anything which increases the rapidity of the pulse increases the rapidity with which chloroform is conveyed to the nerve centres, and if alcohol, or atropine, or caffeine does this they are bad. But alcohol does not always increase the rapidity of the pulse. Very often if a patient is frightened a small dose steadies and slows the pulse, and gives him confidence; and in so far as it does this, a preliminary dose of alcohol does good in operations under chloroform.

Before bringing this lecture to a close, I desire to call your attention to a paper by Dr. James Dunlop in *The Lancet* of September 27th, 1890, as it has an important bearing on Professor Wood's address. Dr. Dunlop's paper consists of observations on the administration and dangers of anæsthetics, in reply to *The Lancet's* circular calling upon scientific medical men throughout the world for information on the subject. Dr. Dunlop states that "the skilful administration of the anæsthetic includes *attention to the pulse*, as well as watching the respiration ", and he gives numerous instances of death under chloroform from what he calls asphyxia and syncope. Dr. Dunlop's paper emphasises the truth of what I have already stated with regard to the worthlessness of Professor Wood's clinical facts, and it affords additional evidence that if part of the chloroformist's attention is devoted to the pulse in chloroform administration, deaths occur. It must be clearly understood what is meant by attention to the pulse in chloroform administration. Any surgeon who can give chloroform safely can keep his eye on the pulse. Towards the end of long operations I often ask my students, just as Mr. Syme used to ask me, "How's the pulse ? " This is quite a different matter from watching the pulse for signs of danger from the anæsthetic—which is both unnecessary and dangerous. It is unnecessary because no reliable information as to the effect of chloroform can be obtained from the pulse, and it is dangerous because if ever the administration of chloroform is pushed far enough to cause the pulse to show signs of failure of the heart, the limits of safety have already been so far exceeded that a fatal result must almost inevitably ensue.

It is strange to find a surgeon of Dr. Dunlop's standing writing of deaths under chloroform occurring from asphyxia. The Hyderabad Commission has pointed out that the only danger of asphyxia, as of holding the breath, in chloroform administration, is that it makes patients gasp and so take in an overdose with extreme rapidity. Dr. Dunlop asserts that deaths from syncope occur early in the administration of chloroform. He says : " A small quantity of concentrated vapour without any air administered to a nervous, timid patient has proved fatal. A healthy florid-faced young man had chlorofrom administered to him in private by a surgeon who had gained much experience of chloroforming in the wards of the Royal Infirmary. The chloroform was administered on a folded towel, three or four drachms having been poured on. While the patient was counting, and when he had reached the number twenty-five, his face became at once pale and blanched, the pupils suddenly widely dilated, and no pulse was felt at the wrist. The young man was dead." In order to understand the cause of the young man's death, you must consider Dr. Dunlop's account of it carefully, and remember that it is impossible to make any patient (or animal) inhale concentrated chloroform vapour without any air, unless you either cause him to gasp, by making him hold his breath or by asphyxiating him, or first render him insensible. From Dr. Dunlop's statement it is clear that the young man was made to count while the chloroform was held over his face on a towel. No surer method of making him gasp could possibly have been devised. Try it yourselves without chloroform, and tell me if counting and regular natural breathing are compatible. But with chloroform close to the face, it is certain that if you try to count you must gasp after every number, or after every few numbers. In fact, counting must lead to gasping much more quickly than holding the breath, because the lungs are emptied by the process. You remember a case which occurred here the other day. When a patient was nearly under chloroform he suddenly began to try to speak, and went on muttering "*er—er—er—er—er*" for a long time without taking a breath. I told the student to remove the chloroform cap from his face, as the patient was sure to gasp, and after a few seconds more the gasp came. Fresh air entered the lungs, and the patient recommenced breathing regularly, and very soon went over; but if the cap had been kept near his face, he would have inhaled a large dose of chloroform, and might have been at once poisoned. Substitute counting for the noise our patient made, and you have the true explanation of the cause of overdosing and death in Dr. Dunlop's case. Dr. Dunlop says no disease was found in any of the young man's organs on post-mortem examination, and adds: "Strange as it may seem, disease of valves, hypertrophy of walls, cardiac murmurs of all kinds give no trouble during the administration of chloroform." According to this teaching, the more healthy a man's heart is the more certain he is to be liable to sudden death from syncope under chloroform; and if this is to become part of our belief, we shall soon have to refuse chloroform to anybody who has not a diseased heart. This is on all fours with the text-book teaching with reference to shock under chloroform. It is not the capital operations like amputations which are said to be dangerous from shock under chloroform, but only the trivial ones, like the operation for squint or for ingrowing toe-nail. In order, therefore, to make the operation for squint safe under chloroform, the patient ought first to have his leg off! You may believe me that there is no such thing as chloroform syncope, and that all deaths *from chloroform* are due without exception to over-dose. You never hear of syncope except in the practice of those who do not know how to give chloroform with safety; and when once the truth is accepted, as it must be eventually by the whole profession, chloroform syncope will disappear into the region whence it came.

It is now time to ask what are the results of *The Lancet's* call for clinical experience. It seems to me that sufficient evidence of the most crushing kind has been accumulated in *The Lancet* for 1890 to show that if the chloroformist's whole attention is not directed to the respiration, so as to keep it regular, deaths are inevitable. First, we have had the statistics of Mr. Roger Williams (vide *The Lancet*, Feb. 8th, 1890), to show that one patient dies in every 1,236 cases of chloroform administration at St. Bartholomew's and the other large London hospitals where the pulse is taken as a guide. Then we have Professor Wood's statement that "carnage" still goes on when chloroform is given in accordance with his own principles ; and now we have Dr. James Dunlop asserting that the pulse must be attended to as well as the heart, and boasting that *only three cases* are killed by chloroform in Glasgow alone every year.* Why are we to continue to study only the experience of those surgeons who cannot give chloroform safely ? Why does not *The Lancet* ask the men who do not have deaths to give us their experience for a change ? It is imperative that, in addition to clinical experience of the latter variety, *The Lancet* should call for the opinion of the whole profession on the anæsthesia controversy. The report of the Hyderabad Commission was published in the leading medical journal last January, and many letters and criticisms on it have appeared since. A careful study of *The Lancet* for 1890 will enable every general practitioner in Great Britain to form an impartial opinion as to the merits of the case. The votes of the majority will then decide whether the teaching of the Hyderabad Commission or that of Professor Wood is thought to be more likely to conduce to the interests of the profession and the public ; and I have no fear what the verdict will be.

Finally, Dr. Dunlop states that "every intelligent assistant I have had *during the last ten years* has felt happier during an operation when ether was used than when chloroform was administered." This is not at all surprising. No assistant can give chloroform happily or safely if he is always in dread that his patient may die suddenly. As long as the belief prevails that one of the dangers of chloroform is sudden death by stoppage of the heart, and the chloroformist watches the pulse for signs of heart failure, deaths will occur. It is high time this reproach to our profession and opprobrium of modern surgery should be put an end to ; and if surgeons will not put an end to it themselves, the administration of chloroform should be prohibited by law until the teaching with regard to chloroform syncope is altered and brought into conformity with the conclusions of the Hyderabad Commission.

## LETTER FROM SURGEON-MAJOR P. R. GABBETT.

### TO THE EDITORS OF "THE LANCET".

SIRS,—Having noticed in your columns several letters and addresses on the administration and effects of chloroform, and the diversity of opinion that appears to exist on this important question, I should like to record what I have myself witnessed during the past eighteen months. By the kind permission of Surgeon-Major Lawrie, the Principal of the Hyderabad Medical School, I have been present at numerous and severe operations performed by that surgeon at the Afzul Gunj Hospital ; and during this period I have never seen the slightest ill-effects result from the administration of chloroform, though sometimes in abdominal operations the patient has been kept under the influence of the anæsthetic for from an hour and a-half to two hours. It has always been given by one of the native students of the school, either male or female, apparently with the greatest confidence and ease on their part, following the principle taught by Surgeon-Major Lawrie, namely, to concentrate the whole of their attention on the breathing of the patient, and not to pay the slightest regard to the heart or pulse. I understood that it had been clearly and satisfactorily proved by Dr. Lauder Brunton and the Hyderabad Commission that chloroform did not prove fatal by sudden stoppage of the heart; yet very little attention seems to have been paid to the addresses of Dr. Lauder Brunton or Professor Wood at the Berlin Medical Congress last August, when the views expressed by these gentlemen were distinctly opposed to each other. The profession in general are therefore left as far as ever from a sound and definite basis for the administration of chloroform.

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* It is fair to Dr. Dunlop to state that he is the apologist of others, and that in some of the deaths under chloroform which he reports the respiration *is believed* to have been watched, and not the pulse. It is evident, however, that it is no good to watch the respiration in chloroform administration if disaster is deliberately courted by the adoption of a plan so fraught with danger as making the patient count.

I am, Sirs, yours truly,

P. R. GABBETT,

Secunderabad, Deccan.

Surgeon-Major, Medical Staff.

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## ARTICLE BY Dr. DUDLEY W. BUXTON,

[*Reprinted from "The Lancet" of December 13th,* 1890.]

One of the dangers of controversy is the almost inevitable crystallisation of the thought of those who advocate, or press, the negative of any question. There is certainly some fear in the present juncture, lest the common-sense side of the chloroform question should be left out in the cold, while we hotly discuss the *modus operandi* of its death-dealing properties. On the present occasion I hope to draw attention as concisely as possible to the various practical issues involved in my title. Chloroform, I take it, cannot, even if we would, be laid aside; it possesses properties so valuable and so useful that it will, perhaps for ever, remain a favourite anæsthetic with many operating surgeons and obstetricians. The main considerations, then, for practical men are: How far is it a dangerous agent, in what does its danger consist, and can this danger be abrogated or minimised so as appreciably to render the peril beyond ordinary computation either by methods of administration or other means; and, lastly, how does it compare, as far as absolute safety goes, when placed side by side with other anæsthetics?

It may be said that the present time is hardly ripe for opening such an inquiry, since the subject is still to be further elucidated by the clinical report promised us by the Editors of The Lancet. But we should remember that from its very nature such a report must be devoid of personal colour, being statistical, so that the experiences of practical anæsthetists can neither forestall nor be devoid of value whatever may be the finding of The Lancet Chloroform Inquiry. Death under chloroform is, unhappily, a frequent occurrence. We hear of a few deaths, probably not all, and if we accept 1 in 3,000 as a working estimate of their frequency we are probably within the mark, although Surgeon-Major Lawrie says he has given chloroform five or ten times daily for fifteen years without a casualty, *i.e.*, a mean of 40,000 anæsthetics ; and other exceptional estimates have been given by Baudens, 1 in 10,000 during the Crimean War, and Hunter McGuire, 1 in 15,000 during the Secession War. These deaths may be due to—(1) impure chloroform ; (2) careless anæsthetisation ; (3) careful but faulty methods —in any of which events the chloroform is not to blame ; (4) the direct effect of the anæsthetic, due to its intrinsic properties, and unavoidable by care, skill, or the most approved methods.

No. 1.—The impurities of chloroform which may produce untoward effects are free acid, free chlorine. These, however, always give rise to coughing, and assail the nose of the administrator as well as the patient. Chloroform, when left for long exposed, liberates noxious fumes, and should in this case not be used ; and chloroform, when given in a room heated strongly by illuminating gas, also decomposes, and most pungent and irritating fumes are given off, but only the utmost carelessness could fail to detect such a state of things. I think we may take it that by far the greater number of cases of chloroform deaths occur in public institutions, hospitals, &c., where the drug is in constant use and for several patients. We may fairly say then that reason No. 1 accounts for but few cases.

No. 2.—Is probably a more common source of danger, and is one which is likely to be still more prolific of evil if the belief gets abroad, as a daily paper expressed it after the first authoritative statement, that the heart was unaffected by chloroform, that taking this anæsthetic possessed no more danger than taking a glass of whisky-and-water. If the contention of those who deny primary cardiac syncope as a possibility in chloroform narcosis be just, any method which ensured due respiration and a percentage of chloroform below 4 per cent. must be approved and be safe, unless the personal equation of the administrator coming in changes the safety to danger by his failing to watch the respiration, &c.

No. 3.—Undoubtedly some methods are open to grave cavil. but by far the most common methods employed are the open method, *viz.*, the use of lint or a towel, Skinner's mask or Junker's apparatus, and these are not faulty in principle, although dangerous in that the administrator may through heedlessness lessen or increase the strength of the anæsthetic to a dangerous extent.

No. 4.—Chloroform kills, all admit, through respiratory paralysis ; while observers are divided as to whether it also (α) allows death through reflex shock in a patient partly or wholly under its influence, or (β) destroys life by direct and primary heart syncope. Admitting only that extinction ensues through respiratory paralysis, one party affirm, and so far justly if we admit their premisses, that the duties of the administrator are really free from anxiety ; watch the respiration, say they, and if it fails take your measures promptly—artificial respiration undertaken before the heart has given out (secondary heart syncope) will lead to resuscitation. On the other hand, those who contend that chloroform kills directly through the heart teach us a mournful story. Take the utmost care, use the most perfect method in the most approved and conscientious fashion, watch for the inception of symptoms of danger and grapple promptly with the peril, and yet in spite of all a certain number of the patients will die. Both will admit the danger from heart syncope secondary to respiratory failure, and both will submit that such deaths should not occur; that they mean in many cases carelessness and deviation of the anæsthetist's attention from the patient, and both will assert that most of such patients can be brought out of their peril by prompt and skilful employment of artificial respiration, &c. It then becomes necessary to consider the evidence upon which these rival schools of thought and teaching base their opinion and practice. In this connection I would point out a most valuable source of information which has up to the present been but lightly laid under contribution, and which is capable of furnishing the most valuable testimony. I refer to the frequent cases which are met with by those who have a large practice in chloroforming in which untoward symptoms occur, but which not ending fatally are not recorded in the medical press, and perhaps noticed only by the chloroformist himself.

Upon the question of primary heart failure the Hyderabad Commission have definitely assumed the position that respiration always fails before cardiac syncope; in other words, that cardiac syncope is invariably secondary. The immense care with which the work of the Commission was conducted, the great help it received from the experience of Surgeon-Major Lawrie in chloroforming and that of Dr. Lauder Brunton in physiological research, render its conclusions of especial value, and justify us, I think, in saying that if primary heart failure from chloroform is a delusion the Hyderabad Commission were competent to prove the same up to the hilt. The evidence adduced is now common property, so that detailed quotation is needless. In chloroforming considerably under one thousand dogs, rabbits, &c., the Commission failed to induce primary syncope, and Surgeon-Major Lawrie in, say, 40,000 inhalations in human beings the bulk of whom we may assume were Asiatics, never lost a patient. Two classes of fatalities occurred among the lower animals—(1) death from failure of respiration, and (2) accidental deaths, the cause of which the report fails to recognise, except that it assumes it was due to an overdose. An effort was made to bring about fatty changes in the dog's heart by giving phosphorus, &c., but even then primary heart failure did not occur, nor were the Commissioners able to show any evidence of shock occurring in the lower animals whilst under chloroform. If this brief statement fairly represents the outcome of the Hyderabad Commission's work, we find their evidence to be wholly negative upon this important matter. But the question of primary heart failure does not rest solely upon the negative evidence mentioned above. Snow and the older authorities fully recognised the condition; and the Glasgow Committee, both in their original and recent reports, have distinctly asserted that even among the lower animals primary heart failure occurs, while it has been left for Professor MacWilliam of Aberdeen to explain the *modus operandi* of the prejudicial action of chloroform upon the heart. Again, the independent testimony of Professor Wood of Philadelphia has been given to the occurrence of primary heart failure under chloroform, and quite recently he had been at the pains of re-investigating the matter, with a result confirmatory of his original decision. Reverting to the careful research of Professor MacWilliam, we find the following phenomena narrated and graphically recorded by an ingenious arrangement of apparatus. Animals kept under artificial respiration worked by an arrangement which ensured equal and tranquil breathing, and at the same time enabled the operator to give any definite percentage of chloroform, were watched to see what effect the chloroform so given had upon the heart. This effect is notable. The heart muscle relaxed, and dilatation took place, leading, of course, to gradual failure of circulation. Up to a certain point this "give" of the heart muscle was recoverable, and upon chloroform being stopped the heart regained its normal tone and contraction. Other phenomena associated with the action of chloroform upon the heart are summed up under the term "delirium cordis," or a rhythm of the heart muscle leading to futile contraction of portions of the myocardium without assisting the viscus to effectually contract and expel its contents. In some cases the dilatation was sudden, and all efforts at resuscitation failed. These results are important, in so far as they are positive, not negative, and because they are demonstrable; the heart is seen to undergo dilatation and to contract up to its usual capacity, or to give out altogether, as the case may be. We have, then, to a certain extent, a conflict of opinion—the one based upon negative, the other upon positive evidence, at least in the field of experimentation; but I submit neither view can be allowed to go wholly unquestioned when we pass from dogs and monkeys to men. It seems consistent to examine how far the one or the other opinion is borne out in our experience in hospital and in private practice. I find that in most hospitals the directions are carried out which the Committee of the Royal Medico-Chirurgical Society promulgated in 1864, and which are practically those re-asserted in 1889, by the Hyderabad Commission, *viz.*, recumbent posture, loosened dress, &c., and that the pulse is watched,—an injunction which the more recent Commission strongly repudiates. And yet I find deaths constantly occur, and those deaths arise, we are told by the chloroformists, from primary heart failure. Respiration, it is asserted, does not fail first. It is competent for a dispassionate commentator to say that the hurry and excitement of a chloroform death are such that mistakes are easily made, and that it is a well-known fact that the thorax works as if in respiration even when no air can enter the lungs; but, granting this, we have to remember two facts. The "first aid" to those in danger from chloroform is now widely known: the tongue is dragged out, and artificial respiration is started as soon as danger threatens. The other fact is that when the danger is due to primary respiratory failure these measures restore life; when due to primary cardiac failure, they fail. Do most of the cases of peril under chloroform recover or succumb?

Sir Joseph Lister, in his classical article on chloroform in "Holmes' System of Surgery" tells us that he has saved more than one life by directing attention to respiratory difficulties under chloroform. But chloroformists are not so careless as to neglect the elementary law of the anæsthetist, *viz.*, to permit nothing to distract his attention from his patient. Again, the fact that Snow and Clover each met with deaths proves that such events, however they may be lessened in number by skill and care, cannot be reduced to a vanishing point. A further consideration which I am able to adduce from my personal experiences is this, that of the very large number of patients to whom it has been my duty to give chloroform, or the substance called methylene, a certain percentage have given me anxiety. My plan is to use Lister's open method or Krohne and Sesemann's recent and useful modification of Junker's inhaler, the flannel cap face-piece of which permits a free current of air to pass through it. I also make it a rule to test the vigour of respiration by placing my hand over the mouth, to watch the colour of the patient's face and ears, and to keep a finger upon an artery, always testing the initial rate and force of the pulse in that artery at the commencement of the operation. Proceeding in this way I have sometimes found that the heart flags even when respiration, judged by the eye and the plan above mentioned, shows no alteration; that not only is there evidence of general feebleness of circulation, which might be attributed to the fall of blood-pressure, regarded by the Hyderabad Commission as protective, but that distinct and unmistakable cardiac enfeeblement occurs. Further, this is progressive, and likely, it has appeared to me in more than one case, to end in cardiac syncope unless prompt and vigorous measures were used. Inversion, which undoubtedly would be highly prejudicial in chloroform, danger due to respiratory paralysis or asphyxial conditions, proves highly efficacious in such cases of cardiac and circulatory enfeeblement. In contending that a fall of blood-pressure is protective, I think a most important factor has been left out of consideration. For granted that such a fall leads to lessened intake of the narcotic vapour, it also determines a lessened output of that which still circulates in the blood. Things being equal, I believe that chloroform acts more harmfully upon poorly oxygenated blood and tissues, *e.g.*, the anæmic, the cyanotic those whose circulation is depreciated by fatty changes in their heart and other viscera, than upon those better supplied with oxygen. On the other hand, cases present themselves when, from the nature of the operation, the chloroforming is complicated by partial asphyxia! and these offer a contrast in that their danger reveals itself in respiratory difficulty leading even to cessation of breathing. I have found, however, that such cases responded readily to artificial respiration, and danger was over. Words give a poor idea of the contrast presented between these two categories of cases, but one has only to be brought face to face with them to be convinced that their difference is one of kind and not one of degree. Again, having once seen the heart relax and dilate under chloroform, as in Professor MacWilliam's experiments, one recognises the anatomical counterpart of the procession of events which one had encountered again and again in the operating theatre. It would subserve no useful purpose to attempt to explain the discrepancies which appear between the experiments undertaken by the Hyderabad Commission and those of other observers, or to reconcile these results with those arrived at by the daily observations—now hundreds of thousands in number—made by medical practitioners in surgical cases, but it may be mentioned that it has been amply shown, by experience with the lower animals and man, that the more highly organised and differentiated nervous systems are more easily affected by reflex shock. Fainting caused by emotion, common in man, is not a daily occurrence among dogs or monkeys.

It may be remarked that it is singular that Professor MacWilliam's dilatation was not discovered by the Hyderabad Commission. The conditions, however, were wanting, for their animals were permitted to die or get *in extremis* through respiratory difficulties so that stress of the anæsthetic was made more evident in most cases *quâ* the respiration than *quâ* the heart, although some of the cases must undoubtedly have revealed the condition had it been sought. A further point noticeable about dogs is that they are peculiarly susceptible to respiratory failure under chloroform, and in performing physiological experiments upon them the utmost care has to be used unless artificial respiration be employed to prevent death from cessation of natural breathing.

Under the last heading I inquired what conclusions practical men must arrive at when comparing chloroform with other anæsthetics, and here I will not press the point beyond instituting a rough-and-ready comparison between chloroform and ether, when both are administered by skilled persons employing good methods ; and I am bound to say that ether, whatever may be urged against it, has fewer deaths to account for than chloroform. It has been said that chloroform kills at or during the operation, whereas ether kills afterwards. This is not my experience. One's kind surgical friends usually keep one well informed of untoward results following or traceable to the anæsthetic, and yet I have not found ether after-effects more severe or dangerous than those arising from chloroform. The subject of this communication is the administration of chloroform, so that I will dismiss this ether digression, and conclude by inquiring, Where does the above reasoning concerning chloroform leave us? I submit there exists a considerable bulk of direct evidence pointing to the truth of the existence of primary heart failure under chloroform, due to a distinct anatomical result of the anæsthetic, and that disregard of the pulse must tend to induce the anæsthetist to overlook the impending danger, while a careful watch over the colour and pulse, on the other hand, would give timely warning for measures to be adopted which would stave off the peril. Respiration must, of course, also be noted, as death may arrive through that channel. Skill and a lively sense of responsibility, which is not fear of chloroform, will do much to lessen deaths from that anæsthetic, but I submit the courage or recklessness begotten of a false sense of security must only end in still further increasing the death-rate from chloroform, already unnecessarily high. While thus contending that chloroform kills through the heart as well as the respiration, I would not be understood in any way to deprecate its use and value in suitable cases. Because we recognise its dangers it does not follow that those dangers assume an exaggerated appearance indeed, the reverse effect is likely to obtain, especially if we at the same time learn how to recognise and guard against them.

Mortimer-street, W.

## ANNOTATION.

*From "The Lancet," Dec.* 27, 1890.

In the course of his presidential address, delivered at the annual meeting of the South-Western State Medical Society of Ohio, Dr. J. C. Reeve reviews with some care the experience of American surgeons concerning deaths under chloroform. Reports of such casualties occurring in the States lead him to tabulate the following as causes of death :—(1) Sudden death during the stage of struggling or excitement, and this form he finds to occur with alarming frequency. (2) Death from respiratory failure. (3) Death from cardiac paralysis, the heart ceasing to beat while respiration goes on. (4) Death when respiration and circulation appear to stop simultaneously. As to frequency, deaths from cardiac failure are said to be largely in excess of those due to respiratory paralysis. Dr. Reeve criticises the results arrived at by the Hyderabad Commission on the grounds that the number of their experiments was too small when as good a record is extant as 1 death in 28,000 administrations, Confederate Army ; 1 in 15,000 (Hunter McGuire). He adds that such records do not represent the true mortality, as some hospitals show as high a rate as 1 death in 200 administrations. Dr. Reeve questions the validity of arguments based upon experiments made upon the lower animals, pointing out that it is a well substantiated fact that drugs do not always affect human beings and the lower animals the same way, *e.g.*, elaterium kills dogs but does not purge them, pigeons bear enormous doses of morphine, and rabbits cannot be killed with belladonna. The weight of clinical evidence alike of England (Snow), Germany (Kappeler), and America, Dr. Reeve avers, is entirely against the Commission's finding that primary heart failure does not occur in chloroform narcosis, and cases are cited in which it is stated the heart stopped before respiration ceased. He concludes by saying: "I protest, in the interest of patients, against the doctrine that chloroform can be administered with absolute safety;" and Dr. Reeve insists that such a conclusion can only be arrived at by "ignoring a vast amount of evidence, both experimental and clinical,—evidence which outweighs all theories and all doctrines, no matter whose names may be appended to them." We consistently with our motto, "*Audi alteram partem*," can at present only weigh evidence, and are most willing to receive testimony both for and against the views which have been advanced by Snow and Clover on the one hand and Syme and Lister on the other.

[Dr. Reeve's "vast amount of clinical evidence", like Dr. Dudley Buxton's "considerable bulk of direct evidence pointing to the existence of primary heart failure under chloroform", is worthless evidence of what occurs in abnormal chloroform administration. All reliable evidence supports the dictum of the Hyderabad Commission that normal chloroform anæsthesia is free from risk.]

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## ARTICLE IN *THE LANCET*, MARCH 14TH, 1891.

BY

### SURGEON-MAJOR EDWARD LAWRIE.

SINCE the publication of my clinical lecture in *The Lancet* of Nov. 29th, 1890, I have received numerous letters from Europe and from different parts of India requesting me to draw up authoritative rules showing briefly the method of chloroform administration which experience, based upon Syme's principles and upheld by the Hyderabad Commissions, has shown to be uniformly safe:—

(1) The chloroform should be given on absorbent cotton stitched into an open cone or cap. (2) To ensure regular breathing the patient, lying down, with everything loose about the neck, chest, and abdomen, should be made to blow into the cone held at a little distance from the face. The right distance throughout the inhalation is the nearest which does not cause struggling, or choking, or holding of the breath. Provided no choking or holding of the breath occurs, the cap should gradually be brought nearer to, and eventually may be held close over, the mouth and nose as insensibility deepens. (3) The administrator's sole object while producing anæsthesia is to keep the breathing regular. As long as the breathing is regular, and the patient is not compelled to gasp in chloroform at an abnormal rate, there is absolutely no danger whatever in pushing the anæsthetic till full anæsthesia is produced. (4) Irregularity of the breathing is generally caused by insufficient air, which makes the patient struggle or choke or hold his breath. There is little or no tendency to either of these untoward

events if sufficient air is given with the chloroform. If they do occur the cap must be removed and the patient must be allowed to take a breath of fresh air before the administration is proceeded with. (5) Full anæsthesia is estimated by insensibility of the cornea. It is also indicated by stertorous breathing, or by complete relaxation of the muscles. Directly the cornea becomes insensitive, or the breathing becomes stertorous, the inhalation should be stopped. The breathing may become stertorous while the cornea is still sensitive. The rule to stop the inhalation should notwithstanding be rigidly enforced, and it will be found that the cornea always becomes insensitive within a few seconds afterwards. It is only necessary to add that the patient should be so dressed for an operation that his respiratory movements can be seen easily by the chloroformist. In the climate of India this is not difficult to manage, but it is difficult to manage in the climate of Europe; so that in this respect, and in this respect alone, the chloroformist in England is placed at a distinct disadvantage compared with the chloroformist in India. Proceeding in the above way chloroform never produces any bad effects, and its administration, in any case which is fit for an operation, is entirely free from danger.

[The Hyderabad Commission unequivocally adopts the suggestion of Dr. William MacEwen in the great discussion on Anæsthetics in Glasgow that "every one of the patient's respirations should be registered on the administrator's tympanum."]

I have drawn attention to the danger of taking the circulation as a guide as to the effect of chloroform,* and should not further allude to it but for an article on chloroform administration by Dr. Dudley Buxton in *The Lancet* of Dec. 13, 1890. This article is by far the cleverest that has yet appeared from our adversaries, but Dr. Dudley Buxton's teaching condemns itself. His plan of giving chloroform is—"(1) To use Lister's open method or Krohne and Sesemann's recent and useful modification of Junker's inhaler; (2) to test the vigour of respiration by placing my hand over the mouth; (3) to watch the colour of the patient's face and ears; (4) to keep a finger upon an artery—always testing the initial rate and force of the pulse in that artery at the commencement of the operation."

The first remarkable point in these rules is that no fixed principle of chloroform administration is laid down. The chloroformist may employ indiscriminately the open method or an inhaler, according to his fancy. In the second place, if the chloroformist places one hand over the mouth to test respiration, and keeps the finger of the other upon an artery, as Dr. Buxton recommends, it is difficult to understand how the anæsthetic is to be administered, unless the patient is to do it himself. The severest condemnation of the method, however, is to be found in Dr. Dudley Buxton's own words. He says: "Proceeding in this way I have sometimes found that the heart flags even when the respiration, judged by the eye and the plan above mentioned, shows no alteration; that not only is there evidence of general feebleness of circulation, but that distinct and unmistakable cardiac enfeeblement occurs. Further, this is progressive, and likely, it has appeared to me in more than one case to end in cardiac syncope, unless prompt and vigorous measures were used." This shows plainly that the plan is faulty, if not impracticable ; in fact, it stands self-condemned. There is never any dangerous enfeeblement of the general circulation or of the heart in the method of giving chloroform recommended by the Hyderabad Commission. Dr. Dudley Buxton can readily satisfy himself of the truth of this statement by giving our method a thorough and impartial trial. None of our critics have done this yet, but if one fair-minded man of Dr. Buxton's calibre were to do so, he would infallibly convert himself to our views, and the conversion of the rest of London would follow in time as a matter of course. Dr. Buxton states that " having once seen the heart relax and dilate under chloroform, as in Professor MacWilliam's experiments, one recognises the anatomical counterpart of the procession of events which one has encountered again and again in the operating theatre." This statement goes far to prove that there must be some fallacy about Professor MacWilliam's experiments. As a surgeon I care very little what happens to the heart when it is subjected to such abnormal treatment as is involved in laying open the thorax and pumping chloroform into the lungs in order to prove that it is directly affected by chloroform. We might just as well pump in boiling water, and then tell old women they are not to inhale steam when they get bronchitis. But it is interesting to be told by such an authority as Dr. Dudley Buxton that Professor MacWilliam's results form the anatomical counterpart of a method of chloroform administration which causes general feebleness of the circulation and of the heart, and there is very little doubt that they do. Professor MacWilliam's premiss that heart failure is not necessarily accompanied by a fall of blood-pressure is manifestly unsound, and his description of rhythmic cardiac relaxation and dilatation, under chloroform, is open to the fatal objection that he has not shown that the movements of his base line, by which they were estimated, were not produced by relaxation of the diaphragm, pushing up the heart through his artificial opening. If his heart dilatation can really occur without fall of pressure, or when chloroform is properly given, it must be a natural condition and free from risk.

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* *The Lancet*, November 29, 1890.

The Hyderabad Commission has proved that there is no such thing as chloroform syncope, and that in death from an overdose of chloroform the respiration always fails before the circulation. What the harmless fall of blood-pressure in normal chloroform administration is due to, as well as what happens to the heart after the respiration fails from overdosing, which are the only two points left open to discussion by the Hyderabad Commission, must be entrusted to physiologists to determine. In the telling words of Dr. Bomford, " it is sufficient for us as practical men to know (1) that the heart is the very last organ to give in under the action of chloroform, and (2) that there is no more danger of permanently paralysing it in chloroform administration, than there is of paralysing the legs and giving the patient paraplegia."

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## LETTER FROM Dr. DUDLEY W. BUXTON,

### To the Editors of " The Lancet."

Sirs,—Surgeon-Major Lawrie's personal reference to me calls for a brief reply. My paper was not intended to instruct in the elementary methods of administering chloroform, so I omitted details which I thought were familiar to all who had mastered the subject of chloroform administration, and were not so wedded to one method as to allow themselves to remain ignorant of all others. The open method, or that of Syme and Simpson, is so well and fully set forth in Sir Joseph Lister's article in "Holmes's System of Surgery," written in 1861, that it appeared to me to be superfluous to describe it in the columns of a journal like *The Lancet;* but since Surgeon-Major Lawrie has received numerous letters from Europe concerning it, and has gone to the pains of describing it, I suppose I am wrong in supposing that the ABC of the chloroformist's duties are pretty generally understood.

With regard to the second method to which I alluded, that in which Junker's inhaler is employed, I must say that your correspondent is clearly wholly unacquainted with the method, or he would not have permitted himself to indulge in such playful criticism. His humour loses none of its point when read by a person who is familiar with Junker's apparatus. I may just say that, with average intelligence and common-sense, the inhaler can be used without necessitating Surgeon-Major Lawrie's suggestion being carried into effect, and the patient compelled "to do it himself." Further, the argument which he adduces, *viz.*, that because I had seen various degrees of heart failure and circulatory trouble, therefore my methods are erroneous, is an instance of what logicians call a *petitio principii*, and proves too much. In the first place, the open method, to which my remarks mainly referred, is precisely the method which the Hyderabad Commission, following Syme, advocates. I as well as many others in Europe, learnt the method before the session of the Commission. That Surgeon-Major Lawrie has never seen the heart fail and that I have is not surprising, because he has never felt the pulse or examined the action of the heart during chloroformisation, and I have. That Surgeon-Major Lawrie has had many cases I do not for a moment doubt, only he has overlooked them, and as, fortunately, in most cases respiratory rhythm is affected *pari passu* with cardiac enfeeblement, he has seen the danger-signal *quâ* the respiration, and has taken measures accordingly.

I am further stated to give no fixed principle of chloroform administration, and in my paper, to which reference has been made, I admit such is the case, and for the reason above stated, that my object was argumentative rather than didactic. In my lectures and practical demonstrations I believe I am dogmatic enough, but personally I regard dogmatism in discussion upon scientific subjects in a scientific periodical as unseemly and valueless. In conclusion, may I draw attention to a sentence against which I am bound to enter a protest? It runs: "The Hyderabad Commission has proved that there is no such thing as chloroform syncope, and that in death from an overdose of chloroform the respiration always fails before the circulation." I submit the Hyderabad Commission has done nothing of the kind; its conclusions were based upon purely negative evidence, and were not warranted by the facts before the profession, and I am strongly of opinion that any teaching which tells chloroformists to ignore the pulse is fraught with danger. I am greatly obliged to Surgeon-Major Lawrie for his courteous reference to myself, and can assure him that I constantly employ the open method and obey the rules he, following Syme, advocates; but I also watch the pulse.

I am, Sirs, your obedient servant,

DUDLEY W. BUXTON.

Mortimer-street, Cavendish-square, W.

## LETTER FROM SURGEON-MAJOR EDWARD LAWRIE,

### TO THE EDITORS OF "THE LANCET".

SIRS,—Dr. Dudley Buxton cannot be allowed to depart from his original statement. He described his method of giving chloroform in the following precise terms (the italics are mine): "*My plan* is to use Lister's open method or Krohne and Sesemann's useful modification of Junker's inhaler. I also make it a rule to test the vigour of respiration by placing my hand on the mouth, and to keep a finger upon an artery." It is this plan which I ridiculed. Dr. Buxton now dexterously tries to make it appear that my criticism was directed against Junker's inhaler, and that he gives chloroform on Syme's principles, though Syme never took the pulse as a guide. Dr. Dudley Buxton's plan is impracticable, and its absurdity is only surpassed by the absurdity of the method of another specialist, Mr. Rickard W. Lloyd. Mr. Lloyd proceeds to give chloroform* "with the sprinkled lint lying on the separated fingers of my right hand in front of the mouth, which enables me to feel the force of expiration, with my left middle finger on the left temporal pulse, and my left thumb holding up the left upper lid of the patient and testing the corneal reflex when necessary." Mr. Lloyd thus endeavours to obtain information as to the state of the patient from three if not four distinct tactile impressions at the same time, which is just as dangerous as it would be for one of my students to administer chloroform to three or four patients all at once. Dr. Dudley Buxton further states that I must have had many cases of heart failure under chloroform which I have overlooked, because "Surgeon-Major Lawrie has never felt the pulse during chloroformisation." The whole of this statement is a gratuitous invention. All my operations are performed in public; I have never had a case of heart failure, and I frequently take the opportunity of demonstrating that the action of the heart and the pulse are invariably regular when chloroform is properly administered. I do not allow the pulse to be taken as a guide as to the effect of chloroform, because I know it is no less useless than dangerous.

Finally, Dr. Dudley Buxton asserts that the Hyderabad Commission has not proved that there is no such thing as chloroform syncope, because "its conclusions are based upon purely negative evidence." If Dr. Buxton were as familiar with the A B C of logic as he would have us believe he is with the A B C of the chloroformist's duties, he would know that proof of the absence of anything must always be negative proof. The rest of Dr. Dudley Buxton's letter consists of a bald statement of creed, unsupported by any explanatory reasons, and does not call for further notice.

I am, Sirs, your obedient servant,

EDWARD LAWRIE, Surgeon-Major.

May 11th, 1891

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* *Vide* THE LANCET, April 4th, 1891.

## LETTER FROM DR. DUDLEY W. BUXTON,

TO THE EDITORS OF "THE LANCET".

SIRS,—Together with other earnest seekers after truth, I much regret the tone and lack of courtesy in Dr. Lawrie's reply to my letter upon this subject. Dr. Lawrie will hardly strengthen his position by accusing me of "gratuitous invention," or by the sneer conveyed in his sentence: "If Dr. Buxton were as familiar with the A B C of logic as *he would have us believe* he is with the A B C of the chloroformist's duties, he would know that proof of the absence of anything must always be negative proof." The italics are mine. Unfortunately for Dr. Lawrie's argument, he quotes my words, which are "its (*i.e.*, the Hyderabad Commission's) conclusions are based upon purely negative evidence." I cannot suppose Dr. Lawrie can confuse the terms "evidence" and "proof" or the phrases "negative evidence" with "negative proof." If he does, I may perhaps be excused if I declined to accept his views upon the rules governing the constitution of the logical syllogism. Dr. Lawrie again indulges in calling what he regards as my "methods" by hard names; but this I can forgive, for he evidently does not understand in what they consist. Unless purely personal matters are excluded in discussing questions, the foundations of which are purely scientific, I must decline to participate in them. It cannot tend to increase our knowledge of the physiological action of chloroform for us to hear that Dr. Lawrie *of Hyderabad* regards Dr. Dudley Buxton's [*of London* !!] method as "impracticable," and its "absurdity only surpassed by the absurdity of another specialist." Such an unguarded manner of expression can subserve no useful purpose.

I am, Sirs, yours obediently,
DUDLEY W. BUXTON,
Anæsthetist in University College Hospital.

MORTIMER-STREET, W., *May* 18*th*, 1891.

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[Dr. Dudley Buxton accuses Surgeon-Major Lawrie of lack of courtesy, and if the accusation were well founded, Surgeon-Major Lawrie would apologise to the whole profession. But the view we take of the matter is this. If specialists publish their plans of chloroform administration, while adversely criticising the Hyderabad Commission, they can have no possible ground of complaint if they are criticised, however severely, in their turn. It argues no discourtesy that we consider and know it to be impracticable to give chloroform safely or properly in the manner described by Dr. Dudley Buxton as "my plan".

Dr. Buxton made a statement regarding Dr. Lawrie's practice which is inconsistent with fact. We have yet to learn that any lack of courtesy is involved in the opportunity Dr. Lawrie afforded Dr. Dudley Buxton of honorably withdrawing this speculative misstatement when he pointed out that it was a gratuitous invention.]

# ARTICLE BY ARTHUR R. CUSHNY, A.M., M.B. (Aberd.)

(FROM THE PHYSIOLOGICAL LABORATORY OF BERN UNIVERSITY.)

"*The Lancet*", *March 14th*, 1891.

THE report of the Hyderabad Chloroform Commission has been read with interest not only in England and America, but also in Germany and Switzerland, where it has aroused widespread discussion and comment. In the latter country especially, which is at present in the throes of a discussion as to the respective merits of the rival anæsthetics, the report of the second Commission was awaited with anxiety and received with much approbation by the numerous advocates of chloroform. On the decision of the Commission becoming known, Professor Kronecker of Bern, whose pupils, Ratimoff and Schemey, had already shown the poisonous action of chloroform on the heart, suggested that I should make a few experiments on the subject. The details of these will be published elsewhere, and I will here state merely the general results.

By the Hyderabad Commission no experiments seem to have been made with chloroform vapour of ascertained concentration, and, as this factor seems to be of considerable weight, I have used an apparatus* in which it could be exactly regulated. This consists of two wash-bottles, one-third filled, the one with chloroform, the other with water. Part of the air inspired by the animal passes through one, part through the other, and becomes saturated with chloroform fumes or water vapour accordingly. The proportion of the chloroform saturated to the moisture saturated air is regulated by two graduated stopcocks (Kronecker's *Schiebhahn*). The two currents unite, and are led by a short tube to the tracheal canula. Expiration is provided for by a small hole in the sheath of the latter, which is covered by a flap of moist goldbeater's skin, to prevent the inspiration of air which has not first passed through the apparatus. In order to exclude all chance of asphyxial complications, and to allow a regular amount of chloroform to be absorbed in a given time, the air was driven through the apparatus by a machine for artificial respiration. The animals used were dogs and rabbits, and the experiments were carried out at ordinary room temperature (15° to 17° C.). The movements of the heart and respiration were recorded by a heart needle and diaphragm lever, and tracings were taken in almost all the experiments. In the respiratory tracing the curves formed by spontaneous respiration were combined with those caused by the artificial inflation of the lungs, but could be easily distinguished, and the moment of cessation of the former ascertained. When this occurred artificial respiration was generally stopped for a few seconds, to see if any spontaneous movements occurred. I may here state that the heart needle was not found to be an accurate index of the heart beat, as in some cases it continued to vibrate after paralysis of the ventricles, owing to the continued pulsation of the auricles communicating a motion to the ventricles through which the needle passed. In all cases, therefore, in which there was any doubt as to the condition of the heart, artificial respiration with air was resumed, and the heart observed directly by opening the thorax. As soon as chloroform was applied to a rabbit (except when the vapour was greatly diluted with air), the animal began to struggle and the respiration became irregular with expiratory gasps, or almost ceased for a few seconds. Very soon the stage of rapid respiration described by Knoll set in. In those cases in which the air was saturated with chloroform (*i.e.*, in which all the air inspired passed through the chloroform bottle), this stage only lasted from twenty to sixty seconds. In cases in which a lower concentration was used it lasted much longer, sometimes for two hours, but in *all* the spontaneous respiration gradually got shallower without getting slower, till it ceased entirely. The condition of the heart at this moment varied with the concentration in which the vapour had been blown into the lungs. Of seventeen rabbits chloroformed with saturated air, in five the whole heart continued to beat, in nine one or both auricles were in rapid motion while the ventricles were still, in one the left auricle alone was paralysed, while in one the whole heart was in diastolic standstill. In rabbits which were anæsthetised with a mixture of chloroform vapour and air, *i.e.*, in experiments in which the stopcock on the water-bottle was partially open, the whole heart was invariably found beating, but the more chloroform the mixture contained the weaker was the heart beat. Even when only four parts chloroform-bearing air were mixed with ninety-six parts pure air the respiration ceased, though only after from two hours to two hours and a-half's exhibition of the drug. With lower concentrations than this I could not obtain narcosis, and did not attempt to find how long an animal could be exposed to the vapour. Ratimoff found that rabbits could be kept narcotised for six hours without the respiration ceasing. Paul Bert, on the contrary, found that dogs died after two hours and a-half's constant inhalation if the narcosis was complete. Several dogs were chloroformed by the same method, and in all of these, however concentrated the chloroform vapour, the respiration ceased, while the heart could still be felt pulsating. A rather higher concentration was required to keep dogs narcotised than was necessary for rabbits.

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* Ratimoff and Schemey used the same apparatus in their experiments.

In regard to the restoration of spontaneous respiration, in both dogs and rabbits, I found that success depended not so much on the length of the interval between the cessation of the spontaneous and the recommencement of the artificial respiration as on the concentration in which chloroform had been given. In dogs inflation of the lungs was successful in all cases. In rabbits, on the contrary, it was impossible to restore life when chloroform-saturated air had been used, however soon after paralysis of the respiration restorative measures were taken. When a half-and-half mixture was used, three out of eleven animals could be resuscitated, and with lower concentrations the proportion of recoveries increased till below 25 per cent. all animals could be restored. In one case, in which a 4 per cent. mixture was used, an interval of three minutes elapsed before artificial respiration was begun, and the animal still recovered. The chances of recovery after paralysis of the respiration, in fact, depend entirely upon the condition of the heart, and therefore on the concentration in which the drug has been used. If, as is the case in rabbits, the heart is very much weakened by very concentrated administration, it is impossible to restore the respiration. If, on the other hand, the heart is comparatively unaffected, as in dogs, or in rabbits in which the drug has been administered in a more diluted form, the animal can always be revived provided the necessary measures are taken within a reasonable time. The essential point is that the drug be given in sufficient dilution to avoid its action on the heart, and if this dilution can be sufficiently provided for, observation of the pulse is not absolutely necessary. Although I cannot agree with the Hyderabad Commission that the heart always continues to beat after respiration ceases, yet the difficulty in maintaining the concentration necessary to paralyse the heart simultaneously with the respiration is extremely great, and I should think that in ordinary chloroform administration such a simultaneous paralysis can never occur.

As, in the great majority of my experiments, death was due to the respiration alone, some experiments were made to find the condition of the medulla oblongata during chloroform narcosis. This question was approached by an examination of the reflexes connected with it. I found that these all ceased before the respiration, and returned (in cases where the animal was revived) some time after spontaneous respiration had set in. The more concentrated the mixture, the shorter the interval between their disappearance and that of respiration. The first to cease to act was the corneal reflex, then that on the respiration from the nasal branch of the trigeminus, then the swallowing reflex from irritation of the soft palate or of the superior laryngeal nerve ; and, last of all, Traube's active expiratory reflex from expansion of the lungs. The reflex in dogs were not so carefully noted as in rabbits, but in general presented no marked differences.

The respiratory centre, as Knoll has pointed out, is affected differently in different stages of chloroform narcosis. At first it is acted on by the reflexes first from the nose then from the trachea and lungs. Then the rapid stage of respiration sets in. This occurs after division of the vagi, and is due to stimulation of the vagus roots in the medulla, and not to stimulation of the respiratory centre itself ; for, if the latter were the case, the result would be, as Marckwald has pointed out, respiratory spasms ; whereas, after section of the medulla above the centre and division of both vagi, chloroform breaks up the resulting spasms into regular respirations exactly as weak tetanisation of the vagi does. Last of all, the centre is paralysed by chloroform, and fails to respond to electrical stimulation of the medulla. The blood-pressure was registered in several experiments in which the drug was administered in weak concentration, and I found that there occurred a slight rise and then a very gradual uninterrupted fall. In very weak form (4 per cent.) chloroform caused no preliminary rise, but a slow fall from the very first, though so gradual was the change in pressure that it could only be seen by comparing the curves at intervals of ten minutes. No such sudden falls of pressure were seen as have been noted by some critics of the Commission, which is perhaps to be explained by the abundant and uninterrupted supply of air to the lungs. I may remark that in those experiments in which the movements of the heart were registered directly, they never showed any sudden change, but a gradually increasing weakness, which culminated in paralysis or passed off, according as chloroform was continued or shut off.

For the sake of comparison a few experiments were made with ether, and the same stages were observed as with chloroform, death occurring equally rapidly if undiluted ether vapour was used, and being due to paralysis of the respiration alone or with accompanying heart paralysis. In one case delirium cordis was observed after a minute and a quarter's inhalation. Rabbits anæsthetised with pure ether vapour could not be revived by artificial respiration. Dogs always ceased to respire while the heart continued to beat, as also did rabbits to which diluted vapour had been given, and the majority of these could be revived. The reflexes appear to behave in the same way as in chloroform narcosis. Ether was borne in higher concentration than chloroform, and the respiration could be more certainly restored. For example, in one case 8 per cent. was given for two hours without killing the animal, while with chloroform 5 per cent. was fatal after one hour and a-half. This fact is the more striking because a very much larger quantity of ether than of chloroform is given in a concentration of 8 per cent. Theoretically, 2·7 times as much ether as chloroform should be carried to the lungs by a given quantity of air, and by actual experiment I found a proportion of 2·61 : 1. In both anæsthetics the action on heart and respiration seems the same, a difference in degree only being perceptible. Ether can be given in greater *quantity* with safety ; but so long as the *proportion* of drug to air is kept low enough either can be used with safety. The concentration necessary to keep rabbits and dogs "under" chloroform is respectively four and eight parts chloroform vapour (at from 15° to 17° C.) to ninety-six and ninety-two parts pure air. Since, however, the concentration necessary to maintain narcosis varied in different species of animals, no inference could be made as to that necessary for men, and my results would have been incomplete had I not been enabled through the kindness of Professor Girard and Dr. Niehans to carry out some experiments in their operating threatre in Bern Inselspital. In these experiments the anæsthetic was given in the same way as in my experiments on animals, with the exception that, instead of a tracheal canula, a Y-shaped glass tube was used, the two ends fitting tightly into the patient's nostrils. Expiration was allowed through the mouth, but inspiration through it was prevented by a valved respirator. The patients in general did not object to the method, and one who had to be anæsthetised again some days subsequently begged that the apparatus might be used again instead of the mask. Narcosis was attained in from five to twelve minutes by a mixture of 15 or 20 per cent. of chloroform, and could be kept up by continued inhalation of 5 to 7 per cent. in children and of 7 to 10 per cent. in adults. Almost no excitement occurred. Throughout the narcosis the face and conjunctiva remained red, and in several cases the concentration necessary to keep up insensibility with retention of the corneal reflex was maintained. With ether adults were narcotised with difficulty, and only after prolonged inhalation. Anæsthesia was afterwards kept up by a concentration of 10 to 15 per cent. Children presented less difficulty, requiring 25 to 30 per cent. to induce insensibility, which continued uninterrupted with a concentration of 10 to 15 per cent. In all cases the usual increased secretion of tears, saliva, and mucus was manifested. The difficulty in inducing narcosis with ether seems to confirm the theory of many observers that ether alone is insufficient for this purpose, and must be aided by a partial asphyxia.

The results of these narcoses may be summed up shortly by saying that, to maintain narcosis with chloroform, a concentration of five to seven and seven to ten parts of chloroform saturated to ninety and ninety-five parts pure air must be adopted for children and adults respectively ; while if ether be used, a concentration of ten to fifteen parts saturated to ninety and eighty-five parts pure air is suitable for all ages. The drug may be inhaled in a slightly higher concentration to induce narcosis, but, as soon as the eyelid reflex disappears, the above proportions should be given. The use of the above apparatus (which will be more fully described in the *Zeitschrift für Biologie*) offers an easy method of regulating the strength of the inhalation, but, if the usual method is adopted; the anæsthetic should be poured on frequently and in small quantities rather than at longer intervals and in largest doses. By the latter method of administration the narcosis is rendered unnecessarily, and often dangerously, deep for a short time, and then quickly passes off; while by the former it is kept at approximately the same depth throughout.

## ARTICLE BY SURGEON-MAJOR EDWARD LAWRIE.

"*The Lancet*", *July* 15*th*, 1891.

THE article on chloroform and ether by Dr. Arthur R. Cushny in *The Lancet* of March 14th, 1891, is a valuable and opportune contribution to the literature of anæsthetics, and is especially welcome to the Hyderabad Commission. Dr. Cushny agrees with us in so many important particulars that it is impossible to regard him as anything else than an ally; and though the cause of the Commission does not require bolstering, Dr. Cushny's experiments and report are the best support we have had yet.

Dr. Cushny agrees with us that the effect of ether is precisely similar to that of chloroform only less intense, and that it is very difficult to produce narcosis at all with ether apart from asphyxia. On the practical question he says, "in ordinary chloroform administration such a simultaneous paralysis of the heart and respiration can never occur," and he further states that "no such sudden falls of pressure were seen as have been noted by some critics of the Commission, which is perhaps to be explained by the abundant and uninterrupted supply of air to the lungs."

On all those points Dr. Cushny is clearly a friend to be welcomed. Where he appears to be wrong is in overlooking the effect of the residual chloroform in the lungs after he has finished pumping chloroform into the trachea. It is obvious that artificial respiration must be much more likely to fail when the air in the lungs is saturated with chloroform than when it only holds a very small proportion of the poison. The Hyderabad Commission succeeded in giving chloroform, by the ordinary method of inhalation, in such a dose that artificial respiration failed to get rid of it quickly enough to save the animal's life. This occurred in dogs; and in dogs, with Dr. Cushny, artificial respiration was always successful. In these cases he must allow that the inspired air was fully saturated, and in all of them every by-stander, including *The Lancet's* chosen representative, Dr. Lauder Brunton, was satisfied that the heart continued to beat after, generally long after, the respiration had ceased. Whether or how far the Hyderabad Commission experimented with dilute chloroform is a matter of no consequence. We tried concentrated vapour, in which alone Dr. Cushny can find any danger. We are quite agreed that extreme concentration is dangerous, but this is so because it leads to the rapid intake of an overdose, and not because there is any action on the heart peculiar to any particular degree of concentration. The Hyderabad Commission's experiments, where chloroform was injected into the large veins of the neck, show that even pure chloroform in the blood does not stop the heart till after it has paralysed the respiration.

It is abundantly proved that diluted chloroform, provided it is not too dilute, gradually causes an accumulation of chloroform in the blood sufficient to produce first narcosis, then cessation of the respiration, and finally death. A higher concentration can only bring about the same sequence of events in a shorter time. The higher concentration cannot have any action on the heart peculiar to itself, unless it be that the more concentrated vapour gives rise to spasm of the bronchioles, or in some other way impedes the oxygenation of the blood, and so, *pace* Professor MacWilliam, affects the heart injuriously. It is impossible that the concentrated vapour can act upon the heart in a mysterious way before it gets into the blood. It is clear also that the harmless dilute vapour must in time accumulate in the blood; otherwise it could not bring about narcosis and cessation of the respiration. But we may well enquire why, when this point is reached, it does not then have the same effect as the concentrated vapour? The truth is that the concentrated vapour interferes with natural respiration and the natural oxygenation of the blood; and in the Hyderabad Commission's report, and in that of Dr. Cushny, it is remarkable that there is an utter want of uniformity in all experiments where normal respiration is interfered with, and complete uniformity in all experiments with chloroform inhaled in the natural way. The concentrated vapour of chloroform cannot be voluntarily inhaled by man or by animals. When a patient or an animal is forced to inhale concentrated vapour involuntarily, or if it is pumped into the trachea, the danger lies in the fact that when it is desired to stop giving the chloroform it is entirely beyond control, and though the administration be discontinued, the discontinuance does not actually commence until the residual amount far down in the lungs has been got rid of. Long before this can be effected, either by artificial respiration or in any other way, the patient may be dead.

Dr. Cushny and the Hyderabad Commission are in complete accord with regard to the practical administration of chloroform, and have the same object in view. That object is the dilution of the anæsthetic to a point consistent at once with full anæsthesia and with safety Dr. Cushny attains this end by means of an apparatus. The Hyderabad Commission attains it with equal certainty by giving the chloroform in such a manner as to ensure normal regularity of the respiration. Lister has demonstrated that the strength of vapour which can be naturally inhaled on an open cone or on a towel is from two to four per cent. With regular breathing, if the number of respirations and the number of cubic feet of air inspired in a minute are known, the dose of chloroform inhaled in a given time can be estimated with almost uniform accuracy. But if the breathing is irregular, the intake of chloroform will be variable and uncertain, and there will be danger in proportion to the uncertainty of the dose. Lastly, if the vapour is concentrated so as to compel the patient to struggle and hold his breath, the risk of overdosing is enormous. Asphyxia ensues and leads to deep gasping inspirations and rapid and, may be, irremediable poisoning. It follows from these considerations that the strongest vapour of chloroform which can be inhaled with regular natural breathing constitutes the safe dilution of the anæsthetic, and that the most trustworthy as well as the most universally manageable guide to safe dilution must be regular respiration. The essential factors in chloroform administration are diluted chloroform and regular breathing, but whatever be the degree of dilution regular respiration alone ensures a measured dose and alone maintains regularity of the heart's action and of the circulation. The sole *raison d'etre* of an apparatus is to determine safe dilution. But as this is definitely determined by regular respiration, and as regularity of the breathing ought to be assured in chloroform inhalation whether an apparatus is employed or not, it is evident that an apparatus is superfluous. Theoretically the Hyderabad Commission has no objection to any forms of apparatus or inhaler, except that no apparatus can ensure regular breathing ; but practically we know that their complicated nature demands some of the chloroformist's attention and may lead to neglect of the all-important respiration. An apparatus is therefore not only superfluous, but introduces an element of danger into the administration. To keep the patient's breathing absolutely regular during the inhalation of chloroform, and to sustain the necessary watchfulness for the warnings afforded by the respiration and by the reflexes whereby overdosing can be avoided, requires the undivided and unceasing attention of the chloroformist.

I trust I have made it clear that the Hyderabad Commission has no intention of criticising Dr. Cushny's admirable work in anything but the most friendly spirit. We are thoroughly satisfied. Dr. Cushny may still think that there is theoretical danger to the heart, but he is constrained to admit that it could not occur in practice. Having got so far, we venture to express a confident hope that he will continue his experiments and observations, and that he will yet see his way to accepting the whole truth.

## THE HYDERABAD CHLOROFORM COMMISSION.

### LETTER FROM Dr. RICKARD W. LLOYD,

To the Editors of "The Lancet".

Sirs,—In his article published in The Lancet of March 14th, Surgeon-Major E. Lawrie, President of the Hyderabad Commission, emphasises the importance of the respiration and totally ignores the circulation, as a guide in the administration of chloroform. Undoubtedly the respiration is essentially important, but my view, after ten years' practical experience of anæsthetics, is that the circulation is also sometimes an important guide to the anæsthetist during an operation, and often most valuable. No method of administering chloroform will ensure the uniform behaviour of all patients during the induction of chloroform anæsthesia. The action of chloroform upon a patient is influenced by mental and physical conditions as well as by disease. The effects of the chloroform upon the circulatory, nervous, and respiratory systems, especially when they are diseased, as well as the loss of blood and the duration and nature of an operation (these being the conditions that have to be met by the practical anæsthetist) influence the respiration in such a way in some cases as to make it expedient to obtain all the information that can be gathered of the extent of exhaustion, the probable power of endurance and the general state of the patient. This knowledge is to be obtained from the respiration, the pulse, the pupil of the eye, the sensibility of the cornea, and the complexion, all of which may be under observation at the same time without discontinuing the chloroform, when it is administered in the manner described by me in a paper read before the West London Medico-Chirurgical Society, and which is fully reported in *The Lancet* of March 14th. The condition of the muscular system, as to relaxation, is also sometimes useful. An anæsthetist who relies on all these sources for information will sometimes be able to continue an administration of chloroform when, if he relied upon the respiration solely, he would feel bound to discontinue it. I have on several occasions had the greatest difficulty in satisfying myself that respiration was in progress at all, especially in connection with some operations upon the skull for depressed fracture or other condition in which the functions of the brain were greatly impaired, and have found the pulse most valuable as a guide in such cases, justifying me in supplying sufficient chloroform to prevent reflex movements of the patient occurring. As long as respiration is unimpaired and there is no cyanosis it may not be necessary to go further for information, but early in the progress of administration the breathing may be accompanied by stortorous noises —not true stertor,—the pupils be dilated or the corneal reflex abolished, and yet the anæsthesia may not be sufficient for operation, and in such circumstances the pulse will be found quick perhaps, and not the slow pulse of complete chloroform anæsthesia. When an operation has been some time in progress the respiration may still be good while the patient may have the appearance of extreme exhaustion, and the pulse will then enable the anæsthetist to judge whether it is necessary to hurry or close the operation, or whether stimulants are imperatively called for. Shallow respiration is not uncommon and, when the respiration only is relied upon, dangerously misleading, often when not appreciated giving rise to sudden and alarming symptoms. In practice each of the guides mentioned is of great service, and without them operators would often be disturbed, and sometimes unnecessarily prevented from completing their operations. The following case throws great light upon the effect of chloroform upon the heart and respiration.

In January, 1891, J. B.——, aged fifty-one years, came into the theatre for radical cure of hernia, and it was my intention to give him chloroform; but on seeing him as his appearance presented venous congestion, I gave him ether. After about a quarter of an hour of the operation there was so much saliva and mucus excited by the ether that it was impossible to keep the patient properly under, and I therefore changed to chloroform, which I administered with extra care, for the reason already stated, *i.e.*, in addition to simply satisfying myself that the respiration was good, I proceeded with the sprinkled lint lying on the separated figures of my right hand in front of the mouth, which enables one to feel the force of expiration ; with my left middle finger on the left temporal pulse and my left thumb holding up the left upper lid of the patient, and testing the corneal reflex when necessary, watching the respiratory movements of the abdomen (which was exposed), the complexion of the face, and the pupil of the eye. After about half an hour I suddenly missed the temporal pulse, and found it was also absent from the wrist, notwithstanding that the respiration continued. After a few moments the pulse returned, and the pharyngeal reflex being present I gave two teaspoonsful of brandy in small quantities, which were swallowed. Then continuing the chloroform, guardedly as before, after twenty minutes the pulse ceased again, and I asked the nurse to give an enema of half an ounce of brandy with water, called the attention of those around to the absence of pulse and the continuance of respiration, which they observed before the respiration ceased, when a little artificial respiration so far restored the patient that the operation was continued and completed in another twenty minutes. During this latter time a very little chloroform only was given when the patient had so far recovered as to call out loudly. The patient afterwards told me he felt nothing of the operation. This patient's heart was, so far as could be ascertained, practically sound. Here then was an operation taking place under chloroform, during which the pulse of the patient undoubtedly failed before the respiration on two occasions, the pulse recovering on the first without cessation of respiration, and the respiration failing on the second occasion some seconds after the pulse, and while it was still not to be felt.

I have endeavoured to show in this letter the importance of the pulse as a guide to the anæsthetist, under some circumstances, during chloroform administration; and it is not my intention to express an opinion as to which of the two,—the respiration or the circulation,—invariably ceases first in death from chloroform, believing, as I do, that it is sometimes one and sometimes the other.

I am, Sirs, yours faithfully,

RICKARD W. LLOYD.

Russell-road, Kensington, W., March 15th, 1891.

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# REPORTS TO THE SCIENTIFIC GRANTS COMMITTEE OF THE BRITISH MEDICAL ASSOCIATION.

## REPORT ON AN EXPERIMENTAL INVESTIGATION OF THE ACTION OF CHLOROFORM AND ETHER.

*Read in Abstract in the Section of Medicine at the Meeting of the British Medical Association at Birmingham, July 1890.*

By JOHN A. MacWILLIAM, M.D., Regius Professor of the Institutes of Medicine in the University of Aberdeen.

[*Reprinted from "British Medical Journal" of Oct. 18th, 25th, & Nov. 1st.*, 1890.]

The following research was systematically commenced in 1888, and has been carried on at intervals since then. The experiments have been conducted upon animals of various kinds, chiefly upon cats and rabbits. The most important experiments were performed on cats, and all the results detailed in this paper are to be taken as being applicable to that animal.

## PART I.

The experiments may be divided into two classes. The first class comprises those experiments in which chloroform was given in the ordinary way by inhalation, while the respiratory movements and the blood-pressure were graphically recorded. This method, in addition to leading directly to certain conclusions, gave results which were largely used for comparative purposes.

The second class of experiments comprises those in which a new method of studying the condition of the heart and vascular system was employed. By this method a simultaneous direct graphic record of the action of the auricles and the ventricles was obtained, alongside of a tracing of the arterial blood-pressure ; precise information as to the state of the different parts of the heart was derived directly from the organ itself. There can be no doubt that blood-pressure tracings alone are entirely unsafe guides as to the strength and character of the heart's action, or even of the ventricular beats. The oscillations in a blood-pressure tracing caused by the ventricular beats do not furnish any sure indications of the condition of the ventricular action. A weak ventricular beat may cause a very large oscillation in the blood-pressure trace, and *vice versâ.* I have shown in a former paper that when the heart recommences beating after a period of inhibition (from vagus stimulation), the abnormally extensive oscillations in the blood-pressure tracing caused by the recommencing beats are often due to systoles that are much weaker than normal. The oscillations in a blood-pressure trace depend upon the amount of blood thrown out by the heart into the aorta, and the conditions obtaining in the arterial system at the time. Hence it is evident that blood-pressure records cannot be relied upon as giving accurate information as to the state of the cardiac action.*

*Mode of obtaining Direct Graphic Records of the Cardiac Action and Blood-pressure Tracings simultaneously.*—The animal to be experimented upon was completely anæsthetised with chloroform in a box, and was then made fast in the usual manner upon a Czermak's holder, a tin of warm water being interposed to maintain the temperature as far as possible. A canula was inserted into the left carotid artery, and connected with a Ludwig's kymograph, so as to give a blood-pressure tracing in the ordinary way. Tracheotomy was then performed, and a canula tied in the trachea, through which artificial respiration was maintained by means of a bellows worked steadily and regularly (by a small motor) at a known rate and in such a way as to supply a uniform amount of air at each stroke. The expired air escaped through side apertures. The rate and extent of the artificial respiratory movements were made to correspond as far as practicable to the rate and extent of natural respiration under chloroform.

The thorax was then laid open, the part of the chest wall over the cardiac region—including the lower part of the sternum—being removed. Ligatures were tied round the ribs before they were cut through, in order to prevent hæmorrhage. The internal mammary arteries were clamped, and when necessary hæmostatics were applied to prevent oozing from bone, &c.. The pericardial sac was then laid open, and the recording apparatus placed in position. The whole proceeding was accomplished with only a very trifling loss of blood.

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* Journal of Physiology, Vol. IX., p. 363.

The recording apparatus employed to register the heart's action was one which I have already described in the *Journal of Physiology*, vol. ix. It is adapted for recording the movements of one auricle and one ventricle simultaneously. Either auricle can be registered; sometimes the one and sometimes the other was used. A thread was attached to the tip of the auricular appendix, and this thread led to a receiving tambour which communicated with a recording tambour; the latter inscribed its movements on a smoked surface. In order to prevent possible displacement of the auricle in the course of the experiment, its dorsal part was steadied by fixing in a clamp the parietal pericardium close to the auricular tissue, care being taken not to include any of the auricular substance in the clamp. The clamp was held firmly in position by means of a brass rod. With such an arrangement each contraction of the auricle causes a pull upon the thread and a movement of the lever of the recording tambour. Hence the contraction and relaxation of the auricle are expressed by up-and-down movements of the recording lever upon the smoked surface. Moreover, distension or dilatation of the auricle causes—by slackening the thread—a change in the level of the recording lever; the arrangement of the apparatus is such that the lever rises. On the other hand, during collapse of the auricle the thread is subjected to increased tension, and the level of the recording lever falls. Thus any continued distension or collapse of the auricle will be attended by a corresponding displacement of the recording lever.

In order to obtain a ventricular tracing a boat-shaped vulcanite trough was first slipped underneath the ventricles, so that the latter had a firm bed to rest upon; the vulcanite trough was rigidly held *in situ* by a strong iron rod. Then a float attached to a light lever was made to rest upon the ventral surface of either ventricle, and this lever was connected with a system of tambours in such a way that any movement of the float was communicated to a recording tambour, and inscribed on the smoked surface beneath the tracing simultaneously written by the action of the auricle. The beats of the ventricle cause up-and-down movements of the float resting on its ventral surface, and these movements are recorded on the smoked paper by an up-and-down movement of the recording lever. Further, any dilatation of the ventricle causes an elevation of the float and a corresponding elevation of the recording lever; and any collapse of the ventricle or diminution of its dorso-ventral diameter is attended by a lowering of the float and a corresponding lowering of the recording lever. Thus while the recording lever indicates the beats of the ventricle by a rapid up-and-down movement (excursion), it also indicates continued distension or collapse of the ventricle by a continued elevation or depression of the level from which it starts on its rapid up-and-down movement (excursion). Distension of the ventricle causes an elevation of the general level or base line of the tracing inscribed by the recording lever on the smoked paper; collapse of the ventricle is attended by a depression in the general level or base line of the tracing.

It is impossible for me to enter fully into the principles and mechanism of this mode of recording the heart's action; it is sufficient for my present purpose to state that this method has been found, by an extended experience of its working in experiments of many different kinds, to give accurate indications of changes in the rate and energy of the cardiac action, and also of changes in the state of the heart's chambers as regards distension or the opposite condition. The results obtained with this apparatus have been tested and controlled by the employment of other modes of registration, for example, by the use of light levers resting on the auricle and ventricle and inscribing their movements directly (without the intervention of tambours) upon the smoked paper. There has been a complete agreement in all important points between the results obtained by the different methods.

It may here be remarked that the amount of mechanical interference involved in the application of the above method does not cause any serious injury to the state of the heart and the vascular system generally. The heart goes on beating with remarkable regularity and steadiness for many hours; a high blood-pressure is maintained, and the circulation is carried on with great activity.

The rate of the movements of artificial respiration is usually indicated in the auricular tracings by a slight rhythmical oscillation in the base line of the tracing. A time record (seconds) is inscribed on the same paper alongside of the other tracings; and, in addition, an electrical signal marked the points of time at which various events (giving of chloroform, &c.) occurred.

Long sheets of smoked paper were employed, and upon them there was obtained side by side, by the method described, a simultaneous record of (1) the arterial blood-pressure, (2) the action of the right or left auricle, (3) the action of the right or left ventricle, (4) the number of the artificial respiratory movements, (5) a time trace marking seconds, and (6) the inscription of an electrical signal indicating the time of occurrence of certain events.

*Mode of Administering the Anæsthetic.*—The bellows employed for artificial respiration were connected with the canula in the animal's trachea chiefly by India-rubber tubing. In the course of this tubing a wide-mouthed bottle was interposed, so that the air coming from the bellows passed through the bottle. The tube leading from the bellows was attached to a glass tube which passed through the cork and reached down to within one-eighth of an inch of the bottom of the bottle. The air entering the bottle through this tube passed out by another tube which projected just beyond the surface of the cork internally, and was connected outside the bottle with a piece of India-rubber tubing leading to the tracheal canula. Between the bottle and the tracheal canula were two side tubes through which a very large part of the air sent by the bellows through the bottle escaped, so that only a small part of the air that passed through the bottle entered the animal's lungs. Through these side tubes the air sent out from the lungs in expiration also escaped.

The anæsthetic to be administered was mixed with the air as it passed through the bottle. This was done by putting the chloroform or ether into a hypodermic syringe, the needle of which was made to pass through the cork; the anæsthetic was then, by means of the syringe, thrown into the bottle in the amount desired. In the case of chloroform, small quantities were used commonly one minim at a time. This amount usually required about twelve (artificial) respiratory movements to evaporate it, and, as each movement of the bellows sent fully 4·54 cubic inches of air through the bottle, it is evident that the relative amounts of chloroform and air were 1 minim of chloroform in about 54·5 cubic inches of air. Now as 1 minim of chloroform, when evaporated, gives only a fraction over 1 cubic inch of vapour, it is clear that the strength of chloroform vapour in the air was about 2 per cent. No doubt, however, the evaporation of the chloroform was somewhat more rapid in the earlier stages than in the later, when most of the chloroform had volatilised and the surface of fluid exposed to the air was diminished ; but, even allowing for this, it is certain that at no time was the amount of chloroform vapour in the air over 3 per cent., and even when more chloroform was added before the first minim had evaporated, the strength was always kept under 4 per cent.

Different samples of chloroform (obtained from Messrs. Morson and Son, Martindale, and others) were used. Each of the samples was carefully tested, and the following results were obtained :—

(1) Appearance and smell characteristic of pure chloroform ; (2) specific gravity 1·497 ; (3) absolutely neutral to litmus paper ; (4) when dropped into distilled water transparent globules were formed, with no milky halo of opalescence ; (5) not coloured on agitation with sulphuric acid ; (6) when shaken with distilled water nitrate of silver gives no precipitate ; (7) no green colour with chromic acid ; (8) iodine gives a violet colour.

When ether (specific gravity 0·725) was used instead of chloroform the amounts injected into the bottle were much larger—15 to 25 minims at a dose.

(For the sake of brevity I shall refer to the bottle in which the anæsthetics were administered as the " anæsthetic bottle.")

## PART II.

*Mode of Conducting the Experiment.*—During the whole progress of the experiments the animal was kept in an unconscious state. In addition to the necessity of obviating any chance of suffering, it was essential for the accurate working of the registering apparatus and the success of the experiment that the animal should be motionless ; the slightest movement or displacement might seriously interfere with the precision and regularity of the graphic records. And of course the use of other drugs, such as morphine or chloral, was contra-indicated, as they would complicate, and might vitiate, the results. The animal was accordingly kept under chloroform during the whole of the time, excepting the periods when ether was given instead of chloroform. Thus the experiments consisted in the administration of a further amount of the anæsthetic to an already unconscious animal, so as to induce a deeper anæsthesia. When the increase of chloroform was about to be given, the condition of the animal was frequently such that it was unconscious and motionless, the heart action and blood-pressure steady and regular, the majority of the reflexes abolished ; but unconscious winking of the eyelid on touching the conjunctiva was commonly present. A further amount of chloroform (usually 1 minim at a time) was then injected into the anæsthetic bottle, and there it evaporated and mingled with the air pumped through the bottle by the bellows. Only a small portion of the air passing through the bottle entered the animal's lungs ; the greater part escaped through the side tubes. Thus only a small portion of the chloroform introduced into the bottle reached the pulmonary air-cells. Meanwhile the state of the conjunctival reflex was closely watched, and it afforded very valuable indications as to the action of the drug. In many cases chloroform was given in sufficient amount to cause a complete abolition of the conjunctival reflex ; but frequently there were changes of a marked character both in the state of the heart and the blood-pressure before the influence of the chloroform was sufficient to cause a disappearance of the conjunctival reflex.

The chief effects of chloroform shown by these experiments I shall now proceed to describe.

*Fall of Blood-pressure.*—The fall of blood-pressure well known to result from the influence of chloroform was seen in the usual forms, slight and severe, gradual and rapid. In some cases the fall was preceded by a slight temporary rise.

*Diminution in the Force of the Heart Beat.*—When the blood-pressure has fallen considerably from the influence of chloroform, there is seen a marked diminution in the force of both the auricular and the ventricular beats, indicated by a lessened excursion (or up-and-down movement) of their recording levers. The height of the curves inscribed on the smoked paper by the systole of the auricle and ventricle is markedly reduced.

Now such a reduction in the contraction force of the heart is not necessarily due exclusively to any direct influence of chloroform upon the organ. For a marked lowering of the blood-pressure from causes not directly affecting the heart, (for example, arterial relaxation) leads to a diminution in the force of the cardiac beat. The fall of blood-pressure involves, among other conditions, a lessened resistance to the discharge of blood from the ventricles and a reduction of the blood supply to the heart itself through the coronary vessels. In such circumstances the cardiac systole becomes diminished in force, as can easily be demonstrated with such a recording apparatus as the one above described. Hence the weakening of the heart's beat under chloroform may be in part attributed simply to the fall of blood-pressure brought about by the drug. But though a certain diminution in the force of the cardiac beat may be explained by the associated fall of blood-pressure, such a cause certainly does not account for the marked depression of the heart which occurs under chloroform, and becomes expressed by a dilatation of the cardiac chambers.

*Dilatation of the Heart.*—When chloroform was given in sufficient amount to abolish the conjunctival reflex completely, and very often before that point had been reached, distinct dilatation of the heart commonly occurred. This change involved both auricles and ventricles right and left. Whether the tracings were taken from the right auricle and ventricle or from the left auricle and ventricle, the results were substantially the same ; more or less pronounced dilatation of the whole organ was clearly present. The general level or base line of the tracings (auricular and ventricular) became elevated to a greater or less extent, indicating an elevation of the portion of the auricular and ventricular walls connected with the recording apparatus. This depended upon a continued increased distension or imperfect emptying of their cavities, causing an increase in the dorso-ventral diameter of the various chambers. After a time, unless the dose of chloroform had been too great, the phase of dilatation passed away ; the parts of the heart resumed their former size ; the tracings sank back to their former level or base line, and resumed the characters they had borne before the chloroform had been given.

These changes were caused over and over again with remarkable constancy in the same animal by repeated doses of the anæsthetic. Dilatation and recovery followed one another with great regularity, as the chloroform was given again and again. The occurrence of dilatation is often remarkable for its suddenness ; its commencement frequently begins to be visible in the tracings within a few seconds (often under five seconds) of the injection of chloroform into the anæsthetic bottle.

In some cases the cardiac dilatation affected the whole organ with tolerable uniformity. In other instances the auricles and the ventricles were unequally affected, sometimes the auricles and sometimes the ventricles being more readily influenced. Occasionally the left auricle became very prominently distended. The distension of the auricles may, in some cases, be in part a secondary change. Their dilatation may be brought about or contributed to by a preceding distension of the ventricles, whereby the auricles are rendered unable to discharge their contents in a normal manner, and so become distended. But in all probability the auricular dilatation is partly a primary change caused by the direct influence of chloroform, and not simply by the obstacle to their emptying themselves offered by the presence of an already existing ventricular distension. The fact that the auricles sometimes become affected much more readily and extensively than the ventricles supports this conclusion.

The cardiac dilatation that occurs from the influence of chloroform is independent of any distinct change in the rate of the heart's action. There is no slowing, or indeed any characteristic alteration of rhythm. The rate of action commonly remains unimpaired, even when all the chambers of the organ have become greatly distended. When the dilatation has become extreme the heart fails in its function at the central organ of circulation. Though it continues to contract rhythmically, its action is feeble and entirely ineffective, and its cavities remain gorged with blood. In this condition evidence of rhythmic movement may still be obtained in the intact animal by passing a needle through the chest wall into the heart (as was done in many of the experiments of the Second Hyderabad Chloroform Commission).

*Relation between Dilatation of the Heart and the Fall of Blood-pressure.*—The occurrence of cardiac dilatation from chloroform is usually associated with a marked fall of blood-pressure. Now, a fall of blood-pressure involves various changes, nutritive and mechanical, in the heart; as already mentioned, there is a diminished resistance to the ventricular systole and a lessened blood supply through the coronary arteries. But it can be clearly shown that the cardiac dilatation above described is not due to the occurrence of a fall of blood-pressure and the changed conditions of heart action dependent on that fall.

The amount of dilatation is not always proportionate to, and does not always run parallel with, the fall of blood-pressure. Sometimes there is a marked lowering of the blood-pressure with little or no cardiac dilatation; while, on the other hand, dilatation not infrequently begins before the fall of blood-pressure has commenced, and, indeed, even during the temporary rise of pressure which sometimes precedes a marked fall. Again, at other times dilatation occurs in marked degree when the fall of pressure is being recovered from, when the pressure has risen to some extent, and is still rising.

Further, cardiac dilatation may, though rarely, be induced in a pronounced degree by the influence of chloroform without any appreciable change in the blood-pressure.

*Causation of the Cardiac Dilatation.*—Many years ago it was shown that chloroform in considerable strength is able in certain circumstances to cause contraction of small arteries; this would lead to an increased resistance to the outflow of blood from the large vessels. Hence, the possible influence of chloroform upon the pulmonary vessels has to be considered in relation to the genesis of cardiac dilatation. It is conceivable that a marked constriction of the pulmonary arterioles and an increased resistance in the lung might, by greatly raising the pressure in the pulmonary artery, embarrass the action of the right ventricle, and lead to its becoming distended,—a change that would be followed, secondarily, by a distension of the right auricle. But in such a condition the left side of the heart would not be dilated, but rather collapsed. Now, we have seen that the whole organ becomes dilated—the left side as well as the right—and consequently the above hypothesis of increased pulmonary resistance—even if there was any proof of its occurrence—is entirely inadequate.

It is clear, from what has been stated, that the occurrence of dilatation is not dependent on changes in the systemic blood-pressure (though usually associated with such changes), nor yet upon changes in the pulmonary circuit ; it is due to some influence exercised by chloroform, either directly on the heart itself or indirectly through the cardio-inhibitory centre in the medulla oblongata and the vagus nerves. The influence of the cardio-inhibitory centre can readily be excluded by section of the vagi. I have frequently cut those nerves, and then repeated the administration of chloroform, with the result that the heart became dilated as before. Moreover, the same happens after the injection of atropine in sufficient amount to paralyse the inhibitory function of the vagus. Hence it is evident that the dilating influence of chloroform is exerted, not through the vagus nerves, but directly on the heart itself. Chloroform acts upon the heart ; it causes a marked depression of the cardiac muscle involving a reduction of its tone relaxation of the cardiac walls, and an impairment of their functional efficiency.

*Periodic Depression of the Ventricular Activity after Chloroform.*—In some animals a peculiar result was observed some time after a dose of chloroform had been given. The sequence of events, as shown by the graphic records, is as follows : A dose of chloroform (1 to 2 minims) was injected into the bottle, and led to a more or less marked fall of the blood-pressure and a certain amount of cardiac dilatation. Those results gradually passed away, the blood-pressure rose, and the heart resumed its ordinary condition. But, while the blood-pressure was rising and the cardiac condition improving, or even after the blood-pressure and the heart had recovered, there sometimes suddenly developed a remarkable change in the ventricular action. The force of the beats became greatly diminished, the ventricles were imperfectly emptied, and they became more or less markedly distended with blood. The curves inscribed by the ventricular beats upon the smoked surface were much reduced in height, and the base line or general level of the tracing was markedly elevated for a time. This condition was soon more or less completely recovered from ; but it sometimes recurred again and again in a very remarkable fashion. In some instances this curious periodic effect on the ventricles was seen to follow the administration of very small doses of chloroform, which had only a comparatively slight primary effect on the blood-pressure and the heart. The auricular action is not affected proportionally to the ventricular change,—indeed, it usually shows only a very slight and gradual reduction.

Such a periodic depression of the ventricles had, in my experiments, always been preceded by a dose of chloroform. It is probably due to a direct influence exercised on the heart ; it does not seem to be affected by section of the vagi. I have never seen any depression of this sort as a result of the administration of ether. Indeed, in more than one instance when the periodic depression was strongly marked and kept recurring again and again, a dose of ether was speedily followed by a complete disappearance of the phenomenon in question. The heart's action improved in a remarkable way ; it became strong, steady and regular.

It may be noted that the occurrence of this great depression of the cardiac action in cats some time after the administration of chloroform, and after partial recovery from its effects, has a curious parallel in the records of some cases in the human subject.

*Effect of Ether compared with those of Chloroform.*—The results obtained with ether have been strikingly different from those following the use of chloroform The ether was administered in the same way as chloroform--by injection into the anæsthetic bottle—but larger quantities were used (for example, 25 minims at a time), and the dose was less gradually given. Instead of being gradually introduced drop by drop into the bottle, it was very often thrown quickly in from the syringe. The condition of the conjunctival reflex was closely watched. Often the dose was given at a time when unconscious winking on touching the conjuctiva was distinctly marked. Then a sufficiency of ether was given to cause a complete abolition of the reflex. The blood-pressure unusually fell to some extent, but the cardiac condition was very different from that seen under chloroform. Instead of becoming markedly dilated as with chloroform, the heart showed, as a rule, no dilatation ; or there was very slight and transient dilatation lasting only a few seconds, attaining no serious proportions, and speedily passing away. The occurrence of even a brief and fleeting phase of slight dilatation was chiefly associated with those experiments where ether was given in large amount with great suddenness. In very many instances there was not the slightest indication of cardiac dilatations. On the contrary, a change in the opposite direction was on several occasions observed—a rapid recovery from the attacks of periodic ventricular depression that have been described as sometimes following doses of chloroform. The difference between chloroform and ether in their relation to cardiac dilatation is strikingly illustrated in the tracings obtained in the way I have already described. Again and again in the course of a prolonged experiment the conjunctival reflex was abolished by the use of ether without any special effect upon the heart, whereas in the same animal chloroform, administered in the same way brought about a marked dilatation of the cardiac chambers—when given gradually, mixed with abundance of air, in an amount sufficient to abolish the conjunctival reflex, and indeed often before that point had been reached. In some experiments the anæsthetics were alternately given, and the characteristic results of the two were alternately manifested with great constancy and precision. Chloroform (always under 4 per cent. of chloroform vapour in the air) was given, leading to partial or complete loss of the conjunctival reflex and pronounced dilatation of the heart. When these effects had passed away and the original condition had been restored, ether was given in such an amount as to abolish the conjunctival reflex ; this was usually attended by no cardiac dilatation. When the effects of ether had disappeared, chloroform was given with the same results as before ; then ether again with its characteristic difference of effect, and so on.

When ether is given with the result of causing a fall of blood-pressure, there is at the same time a reduction in the force of the heart's beat. This reduction appears, as a rule, to be simply a result of the fall of blood-pressure and not due to any special effect of ether on the heart itself. It has been already stated that a fall of arterial pressure due to a diminution in the peripheral resistance, for example, to relaxation of the small arteries, is attended by a reduction in the energy of the cardiac beat ; the heart's force becomes diminished from a cause not directly affecting the heart itself. But, unless the fall of pressure is extreme in its amount and prolonged in its duration—as in fatal hæmorrhage, &c.,—the weakening of the cardiac systole is never so great as to render the organ functionally inefficient ; the heart is always able to expel its content as before and no dilatation occurs.

This is the rule with regard to an ordinary temporary lowering of the blood-pressure—from relaxations of the small arteries—and it seems to be applicable to what occurs in the heart under the influence of ether. The vaso motor centre in the medulla is depressed by the ether ; there is thus brought about a certain degree of arterial relaxation, and the blood-pressure falls. This lowering of the pressure leads to a reduction in the force of the heart's beat. When ether does not reduce the pressure appreciably, it does not cause any weakening of the cardiac systole, or only the very slight and fleeting effect before mentioned ; on the other hand, when ether lowers the pressure markedly, it leads to a marked lessening of the cardiac systole—in all probability as an indirect result, the heart being secondarily influenced through the fall of blood-pressure. There is no evidence that the cardiac weakening is more extensive than is warranted by the fall of pressure ; if the reduction of the heart's force was disproportionate to the lowering of pressure the organ would become dilated—which it does not. In the case of chloroform, on the other hand, there is clear evidence that the heart is directly affected otherwise than merely in a secondary way through the fall of blood-pressure, that the enfeeblement of the cardiac walls is disproportionate to the fall of pressure, and that dilatation is induced in a very pronounced fashion by a special effect of chloroform on the organ itself.

It is evident that these results are subversive of the view expressed by Claude Bernard* regarding the action of chloroform and ether,—a view recently endorsed by the Hyderabad Commission. Bernard said : "As to ether and chloroform, their action is almost the same from a physiological point of view, excepting that there is a difference of intensity in favour of chloroform which will lead us generally to employ the latter substance in preference to ether." But the results obtained in the present research prove that the difference between the action of chloroform and ether is not simply a difference of intensity. There is commonly seen a very striking and important difference between the relative influence of the two anæsthetics upon certain functions. Ether can abolish the conjunctival reflex and induce profound anæsthesia with no appreciable direct effect on the heart ; while chloroform, in causing a less deep anæsthesia—in which the conjunctival reflex is not abolished—may directly cause marked dilatation of the whole heart.

*Relative susceptibility of the vaso motor Centre and the Heart to the depressing Influence of Chloroform.*—The fall of blood-pressure caused by chloroform is due primarily to the depressing influence of the drug on the vaso motor centre, leading to arterial relaxation. Sometimes the fall is preceded by a brief and transient rise ; this is dependent on a brief stimulation of the vaso motor centre prior to the depression of its activity. It has already been stated that when chloroform is given in such an amount as to cause a marked fall of pressure, there commonly occurs at the same time a more or less marked dilatation of the heart. In the healthy animal the latter change does not, in the earlier stages of a fall of blood-pressure, appear to play nearly so important a part in lowering the pressure as does the depression of the vaso motor centre induced by the influence of the chloroform. The pressure is, as a rule, much more readily lowered by the action of chloroform on the vaso motor centre than by its action on the heart. But in the latter stages of a great fall of blood-pressure, when the anæsthetic is given so as to produce a profound effect, the cardiac dilatation becomes a factor of prime importance, and it becomes impossible to raise the pressure by any means affecting the arterial system alone ; the pressure can only be raised by restoring, in some measure, the pumping power of the heart, or by the supplying of some mechanical substitute for this power, for example, by rhythmic compression of the ventricles with the hand.

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* *Leçons sur les Anèsiques et sur l'Aspyxie* (1875), *p.* 101.

The relative susceptibility of the vaso motor centre and the heart to the influence of chloroform appears to vary considerably. Commonly the vaso motor centre becomes markedly depressed before cardiac dilatation is evidenced in any considerable degree. In some instances, however, the cardiac dilatation begins early, before the vaso motor centre has suffered any depression and even while it is stimulated, as sometimes occurs as a primary result of the action of chloroform upon this centre.

Possibly a good deal depends on the state of the heart at the time. If the heart is strong and possesses a considerable reserve of power over and above what is necessary to expel its contents in ordinary circumstances, it can readily be conceived that a considerable amount of chloroform might be taken without causing any evidence of failure, the reserve of power making up for the depressing influence of the chloroform ; meanwhile, the vaso motor centre being acted upon by the chloroform, the blood-pressure would fall. On the other hand, if the heart is, to begin with, just able to perform its function, it is intelligible that a comparatively slight depressing influence exerted on it by chloroform would soon cause it to exhibit signs of dilatation. Many nervously weak hearts possibly come into this category.

*Some Effects of the Falls of Blood-pressure.*—The fall of blood-pressure is, in a certain sense, protective ; it retards the continued access of the anæsthetic to the vital organs. I have frequently been struck with the great resistance shown to the influence of both chloroform and ether in animals where a very low pressure was present from arterial relaxation, due to causes other than anæsthetics, for example, voso motor paralysis. In such conditions large doses of chloroform and ether can be given—mixed with the air entering the lungs—with little or no effect on the heart and blood-pressure. On the other hand, the fall of blood-pressure may become excessive and prove a source of great danger. Fatal failure of some of the vital organs may be determined by the defective blood supply attendant on an insufficient pressure, and this in all probability more readily in certain morbid conditions than in the healthy animal.

*Modes of counteracting the Fall of Blood-pressure in the Carotid Artery caused by Chloroform.*—When a great fall of blood-pressure has been caused by the inhalation of chloroform in the ordinary way, inversion of the animal has been found to exert a slight effect in raising the pressure. But by far the most powerful means of influencing the carotid pressure under chloroform is by applying continuous firm pressure over the intact abdomen or to the abdominal aorta directly. In this way, unless the heart has been directly paralysed by chloroform, a well-marked rise of blood-pressure can be brought about in the carotid and other arteries of the upper part of the body (head, neck and chest), and the circulation in that part is greatly improved in rate and volume. This method of causing a rise of pressure under chloroform has been very frequently tested in the course of my experiments, and is strikingly illustrated in the tracings I have taken. Even when chloroform has been given in an amount sufficient to cause stoppage of the respiration and a formidable fall of blood-pressure, the immediate application of firm pressure over the abdomen or compression of the abdominal aorta has, as a rule, caused a rise of pressure. At the moment of respiratory failure, the heart, though dilated, is, as a rule, capable of acting with some effect, but it soon becomes fatally incapacitated in consequence of the influence exerted upon the already-depressed cardiac mechanism by respiratory and vascular failure: the interference with the nutrition of the heart substance resulting from arrest of the breathing, or dependent on an excessive and continued fall of the blood-pressure, even apart from stoppage of the respiration, must necessarily exert a rapidly destructive influence on the activity of an already embarrassed and depressed organ. At the time the respiration stops, the heart is usually (though not always) able to act in a way sufficient to maintain a certain amount of blood-pressure, compatible with the continuance of life, provided the vaso motor system is not paralysed. The heart then being, in the majority of cases, still able to exercise its function to some purpose, it is easy to understand that a great increase in the peripheral resistance should be attended by a rise in the arterial pressure, the amount of the rise varying largely according to the power still possessed by the heart. The application of firm pressure over the intact abdomen causes, in addition to an increase in the peripheral resistance of the arterial system, the propulsion of a considerable amount of blood from the vessels of the abdominal cavity through the vena cava inferior into the heart. Compression of the aorta by itself causes a great rise of pressure; this shows that a large increase of the peripheral resistance, even apart from the forcing of more blood into the right side of the heart, is sufficient to cause an extensive rise of pressure.

## PART III.

*Relation of the Results described above to what occurs during the Inhalation of Chloroform in the Intact Animal.*—How far are these results applicable to the intact animal inhaling chloroform in the ordinary way? It is, of course, obvious that in certain respects the conditions of experimentation above described are widely different from what obtain when an uninjured animal takes in chloroform by its natural respiratory movements in the ordinary way. In those experiments the thorax was laid open, rendering spontaneous breathing impossible, and artificial respiration by pumping air into the lungs was substituted. With artificial respiration of this kind, although the amount of air and the percentage of chloroform entering the pulmonary air cells may be the same as in natural breathing, still there is a certain difference to be considered, namely, that when air is pumped into the lungs the pressure within the lungs is a positive pressure during the phase corresponding to inspiration (that is, when the lungs are inflated with air); whereas, during the inspiration of air in natural breathing the pressure within the lungs is negative. (It is not probable that this difference is of importance, for there is much evidence obtained by different workers which tends to show that the effects of chloroform depend upon the amount of its reaching certain parts in a given time, the exact mode of its introduction not making any essential difference. But direct proof on this point will be adduced presently.) Further, in the experiments above described, the pericardial sac was laid open, the heart was exposed, and recording apparatus was brought into connection with it.

How far such abnormal conditions may invalidate the results obtained, as far as their application to the ordinary inhalation of chloroform is concerned, has now to be considered. I hold that they do not materially interfere with the applicability of the conclusions arrived at. It seems to me that there is conclusive evidence showing that the action of chloroform, in the respects above detailed, is essentially similar in the conditions under which my experiments have been conducted, and during ordinary inhalation in the intact animal. I shall now proceed to state briefly some proofs of this conclusion.

1. With regard to the possible influence of the presence of recording apparatus upon the cardiac action, it is important to notice that the more considerable changes in the state of *the heart can be verified by direct inspection of the exposed heart without the use of apparatus, though, of course, such a mode of examination is insufficient to detect the finer changes that go on. The broad fact of the occurrence of cardiac dilatation I have often verified by simple inspection of the heart without the use of any recording apparatus. Hence it is plain that this* change is not dependent on any interference with organ from the application of the recording apparatus ; and, indeed, there can be no doubt that when the recording apparatus is used the action of the heart can hardly be disturbed in any serious degree, for it continues to work during long periods with very remarkable regularity and efficiency. Moreover, for comparative purposes (for example, the relative effects of chloroform and ether) the influence of the apparatus cannot interfere, as it is constant.

2. In regard to the opening of the pericardial sac, I shall only remark here that I have been able to observe distinct changes in the heart, of the nature already described, even through the intact pericardium.

3. With reference to the altered respiratory conditions present when the results described in Part II. were obtained, I have made a number of experiments to compare in the same animal the influence of a certain amount of chloroform (*a*) during the ordinary inhalation of chloroform in the intact animal, and (*b*) when air containing chloroform was artificially pumped into the lungs. The method adopted was the following :—A cat, anæsthetised with chloroform, was fastened on a Czermak's support, and the left carotid artery was connected with a kymograph, so as to give a blood-pressure tracing in the ordinary way. The animal was then made to breathe through the anæsthetic bottle, inspiring the air by its spontaneous respiratory movements. This was done by connecting the bottle (*a*) with an air-tight bag tied over the animal's mouth or nose, or (*b*) with a canula inserted into the trachea. The connection between the anæsthetic bottle and the bellows used for artificial respiration was removed. With this arrangement the animal in breathing inspired atmospheric air through

the anæsthetic bottle, through the same channel traversed by the air passing into the lungs when artificial respiration was used. Chloroform could readily be added (by means of the hypodermic syringe) to the air as it passed through the botле : and thus definite doses of the anæsthetic could be administered to the animal, inhaled by its own spontaneous respiratory movements. The effects of accurately measured amounts of chloroform (given in this way) upon the blood-pressure, the conjunctival reflex, and the general condition of the animal were carefully recorded. Then, after an interval, when the effects had been recovered from, artificial respiration was substituted for the spontaneous breathing, the other conditions of the experiment remaining unchanged ; air was pumped through the anæsthetic bottle into the lungs, instead of being inspired in the ordinary way ; an equal dose of chloroform was administered and its effect watched. The influence of the anæsthetic was found to be remarkably similar to what was seen during spontaneous inhalation, provided the rate and depth of the artificial respiratory movements were made to correspond with the rate and depth of the spontaneous respirations. Two or three minims of chloroform induced similar effects, as evidenced by the blood-pressure, the conjunctival reflex, and the general condition, whether the chloroform vapour was taken in by the spontaneous breathing of the animal or was artificially pumped into the lungs. Hence it may be concluded that the intake of chloroform is substantially the same whether it is given by spontaneous inhalation (in which case the pressure within the lungs during the inspiratory movement is negative), or by artificial respiration, when the pressure within the lungs during the phase corresponding inspiration is positive. The abnormal conditions of artificial respiration do not apparently make any essential difference in regard to the effects of chloroform, provided the number and depth of the respiratory movements are the same, and the percentage of chloroform in the air taken into the lungs is identical in the two cases.

4. The nature and extent of the fall of blood-pressure caused by chloroform is, as a rule, an excellent guide to the degree in which the influence of the drug has been exerted upon the general condition of the animal. When the fall is sudden and extensive it gives ground for apprehension that an overdose has been administered ; on the other hand, when the fall is gradual and moderate we may safely assume that the amount of chloroform in the circulation is not too great. Now, applying this test to the experiments described in Part II., we find that dilatation of the heart is present when the fall of pressure is no greater or more sudden than often occurs—without arrest of the respiration or other serious result—when an animal (with intact thorax) inhales chloroform in the ordinary way. For example, in an animal taking chloroform by natural respiration the blood-pressure—as shown by a kymograph connected with the left carotid artery—was reduced by chloroform from 160 millimêtres to 80 millimêtres and again from 160 millimêtres to 94 millimêtres, without stopping the respiration. In the same animal after the thorax had been opened and the cardiac recording apparatus applied, a much slighter reduction of pressure by chloroform—from 112 millimêtres to 94 millimêtres—was accompanied by pronounced dilatation of the heart. Moreover, in one instance, chloroform caused distinct dilatation while the pressure remained tolerably steady at about 106 millimêtres. Such results show clearly that cardiac dilatation may occur at a time when the amount of chloroform in the circulation is not excessive—when there is reason to believe that the anæsthetic is present in no larger amount than during ordinary inhalation.

5. Evidence of great importance in regard to the question under consideration is afforded by the state of the conjunctival reflex.[*] We have seen that pronounced changes may occur in the heart before the conjunctival reflex is completely abolished. This shows plainly that the amount of chloroform in the circulation was not excessive, else the reflex would not have been present. We know that the conjunctival reflex is, as a rule, abolished long before there is any danger of respiratory paralysis. The presence of the conjunctival reflex during cardiac dilatation makes it plain that a comparatively small amount of chloroform—less, indeed, than is commonly used for deep anæsthesia—can affect the condition of the heart in a notable degree. There could hardly be a stronger proof. The abnormal condition of the experiment can scarcely interfere with the abolition of the conjunctival reflex at least not more than operative procedure of various sorts. Further, it is clear that, as a comparative test of the action of chloroform and ether, the presence of the conjunctival reflex is of prime importance. Any influence exerted by abnormal conditions of experiment is present equally in both cases ; hence they cannot interfere with the fair comparison of the two anæsthetics.

6. Another form of experiment may be brought to bear upon the question under consideration. A cat is deeply chloroformed by natural inhalation in the ordinary way, by holding a cloth with a small quantity of chloroform near the animal's mouth and nose. Deep anæsthesia is brought about, the conjunctival reflex being completely abolished, while the respiration goes on regularly. Then the chest is rapidly opened and the heart examined. In some cases I have caused artificial respiration—by means of a canula thrust into the trachea—to be performed as soon as I began to open the chest, in order to avoid any chance of error from an asphyxial condition being developed during the short period elapsing between (*a*) the cessation of the natural respiration (on account of the thoracic wall being cut through), and (*b*) the moment at which the state of the heart became visible. By this mode of experiment I have seen distinct evidence of cardiac dilatation caused by the inhalation of chloroform in the usual way, even though when it had not been carried to such an extent as to paralyse the respiration, and when there were no abnormal conditions to be considered.

7. The evidence obtained from an inquiry into the mode in which chloroform proves dangerous gives information of great importance. Undoubtedly, in the great majority of cases of chloroform collapse, the respiration stops before the heart action has become entirely ineffective. Indeed, in healthy animals, at the time the respiration stops and for a variable period afterwards, the heart, though dilated, is, as a rule, quite able to play its part in maintaining a blood-pressure which, though low, is compatible with the continuance of life. This is, I believe, by far the most common condition in healthy animals. The heart is directly acted upon by chloroform, with the result of inducing a more or less extensive dilatation of its chambers ; but this depression does not, in the majority of cases, assume lethal proportions until the respiration has been arrested. The cardiac change is usually not sufficient by itself to induce a fatal result ; when death occurs, the depression of the heart is commonly associated with vaso motor and respiratory failure. But since the heart is directly acted on by chloroform, and that to a variable extent, it would not be surprising if, in some cases at

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[*] The conjunctival reflex is, in cats, an excellent guide to the general conditions of the animal, though, as in the human subject, there are occasionally slight variations in the time at which it becomes abolished.

least—even in healthy animals—the cardiac change should be, present in a preponderating degree, and should prove the determining factor in bringing about a dangerous collapse; in other words, that death should sometimes be mainly due to cardiac failure, preceding the cessation of respiration. Such I have actually found to be the case in at least three instances, in a total of about seventy animals, where the decisive cause of collapse was the state of the heart. I shall briefly state the main facts of these three cases, in which chloroform collapse was clearly due to cardiac failure, and where the natural respiratory efforts continued to be manifested for prolonged periods after all signs of heart action had disappeared.

Case I.—A cat was rendered unconscious by being put into a box with some chloroform. It was then taken out and more of the anæsthetic was administered in the ordinary way—by chloroform on a cloth held near the animal's mouth and nose. The animal had not been injured in any way, and had not been fastened down, when suddenly collapse occurred. The heart been ceased to be felt and the pupils dilated widely, while the natural respiration went on regularly, though it soon became somewhat slowed and slightly spasmodic in character. Air entered freely at each inspiratory movement. Artificial respiration—by rhythmically compressing the chest—was immediately begun, while pressure was applied to the abdomen. The animal's spontaneous respiratory movements continued to go on regularly, while artificial respiration was also carried on. After this state of things had lasted for five minutes a canula was put into the trachea (compression of the chest being meanwhile continued and the spontaneous respiratory movements still going on), then compression of the chest was discontinued, and air was pumped into the chest from the bellows used for artificial respiration. The spontaneous motions of breathing soon ceased, and a very few minutes later (ten minutes after collapse had occurred) the thorax was opened and the heart viewed through the intact pericardium. The whole organ was seen to be greatly distended, while rhythmical movement of an exceedingly feeble and entirely ineffective character was still seen. The rate of the futile contractions was quick. The ventricles were rhythmically compressed between the thumb and forefinger at about the ordinary rate of the heart beat, and, after a period of fibrillar contraction, the organ recovered its usual mode of action, the pulse was restored, spontaneous respiratory efforts were again manifested whenever the artificial respiration was intermitted, and the whole condition of the animal continued to be most satisfactory throughout the course of a prolonged experiment.

Case II.—A cat, after being anæsthetised with chloroform in a box, was fastened on a Czermak's support; a canula was tied in the trachea, and the left carotid artery was exposed. When the animal was breathing spontaneously through the tracheal tube a little more chloroform was given. The pulse quickly disappeared from the exposed carotid, and the artery became small and collapsed. The respirations did not stop at all, but continued without interruption; they soon assumed a slower and deeper type, and then went on steadily in the same fashion. Pressure on the abdomen, immediately applied, had no effect on the condition of the carotid artery: it remained collapsed as before. Artificial respiration, by rhythmical compression of the chest, was immediately begun and kept up for ten minutes, during which time the spontaneous respiratory movements went on as before. Artificial respiration, by blowing air into the lungs, was then commenced: the thorax was opened, and the heart inspected. The organ was found to be greatly dilated in all its parts, while extremely feeble rhythmic contractions were still present, utterly incapable of discharging the contents of the cardiac chambers. Rhythmic compression of the ventricles was then employed, and kept up for nearly twenty-five minutes, at the end of which the regular action of the heart was restored—more than thirty-five minutes after the collapse had occurred. The ordinary blood-pressure was soon regained, the respiratory centre speedily showed signs of activity, the conjunctival reflex was restored, and the condition of the animal kept up through the whole duration of a long experiment as if no collapse had occurred.

CASE III.—Corresponded in all its details so closely with the history of Case II., that it is unnecessary to relate the sequence of events in detail. The spontaneous respiration continued for ten minutes after the exposed carotid artery had become collapsed and pulseless. Recovery was brought about in the same way as in Case II.

The phenomena presented by all these cases were most closely observed, and recorded immediately afterwards. The features of the collapse showed a remarkable resemblance to what has often been described as occurring under the influence of chloroform in man. Care was taken to leave no room for doubt as to the nature of the spontaneous respiratory movements which went on so long (ten minutes) after the circulatory failure. They were no deceptive movements of the chest without proper entrance of air ; each respiratory effort was attended by a free passage of air into the thorax. The reason why the spontaneous breathing movements continued long even after vigorous artificial respiration was instituted was, in all probability, that though abundance of air was supplied to the lungs by artificial respiration, the collapsed state of the circulation prevented the due conveyance of oxygen from the lungs to the respiratory centre in the medulla oblongata ; hence that centre went on sending out impulses to the respiratory muscles.

It is evident the collapse was not dependent upon vaso motor paralysis ; firm pressure immediately applied to the abdomen produced no beneficial result, as it would have done had the heart not been rendered functionally incapable by the influence of that chloroform.

It is extremely probable that a condition of cardiac failure occurred in some of the experiments of the Second Hyderabad Chloroform Commission. In their Report* (Section 25) they state that it is never in any case certain that artificial respiration will restore the natural respiration and blood-pressure, no matter how soon it is commenced after the respiration stops. And they found that in some cases, even after the spontaneous respiration had been restored, the pressure continued to fall and respiration again ceased ; artificial respiration then failed. Such results were in all probability due to the heart being greatly dilated and unable to perform its functions.

*Occasional Occurrence of slowing of the Heart's Action under Chloroform.*—A temporary but marked slowing of the cardiac rhythm under chloroform I have seen in various intances. Such a change was first illustrated, as an effect of chloroform, by the tracings of the Chloroform Committee of the British Medical Association (Glasgow Committee) in 1879. As far as the evidence afforded by my experiments goes, the slowing of the heart seems to be most commonly due to asphyxial conditions ; the Second Hyderabad Commission has urged that it is always asphyxial. But I have seen facts which indicate that cardiac slowing is in some cases due to causes other than aphyxia, for example, to sensory irritation when the state of the anæsthesia is not such as to render the cardio-inhibitory centre inexcitable by afferent stimuli.

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* *Lancet*, January 18th, 1890.

In any case the cardiac slowing is not due to a direct influence of chloroform on the heart ; it is entirely different in its nature and causation from the enfeeblement and dilatation of the organ described in this paper. The latter, it has been seen, is a result of the direct action of chloroform on the cardiac substance. On the other hand, the temporary slowing of the heart, which occasionally occurs, is due to stimulation of the cardio-inhibitory centre in the medulla, as I found at an early stage of this investigation. It is abolished by section of both vagi, and its characters can be imitated by direct stimulation of the vagi.

Slowing of the cardiac action under chloroform does not, as far as I have seen, prove dangerous in itself in healthy animals, whatever it may do in conditions of derangement and disease. The mode of failure in the healthy animal is not by a slowing or sudden cessation of the cardiac rhythm, but by an enfeeblement and dilatation of the organ while the rhythmic (but ineffectual) contractions go on.

*Relation of the Influence of Chloroform to the Occurrence of Fibrillar Contraction in the Ventricles ("Herz delirium" or Delirium Cordis).*—I have several times witnessed the supervention of fibrillar contraction or delirium cordis in the ventricles of the cat's heart from the influence of chloroform. The normal beat was abolished, and the ventricles were thrown into the wildly-irregular, inco-ordinated, ineffective action characteristic of this condition. But such an accident never occurred in any of my experiments with anæsthetic doses of the drug, but only when an overdose of strong chloroform vapour was pumped into the lungs. I have seen no proof that such a mode of cardiac failure may result from the administration of chloroform in the ordinary way in healthy animals. At the same time it is possible that the occurrence of fibrillar contraction may be a cause of death in some abnormal conditions where the ventricles become prone to assume this disastrous form of activity. The depression and dilatation of the heart induced by chloroform may terminate in a fatal onset of fibrillar contraction or delirium cordis.

## PART IV.

*The Relation of certain Changes in the Respiration to the Effects of Chloroform*—Alterations in the rate and depth of the breathing exert a profound influence on the effects produced by chloroform. The results following the administration of a definite amount of the anæsthetic depend largely upon the rapidity with which it is introduced into the circulation. A small amount of chloroform quickly taken in produces vastly more potent effects than a large quantity slowly absorbed over a considerable period of time. Hence the absolute amount of the drug taken in by an animal—apart from consideration of the time spent in its introduction—proves no guide at all as to the degree of influence produced by it upon the various systems.

A large amount may be given in half an hour with a continuous but slight effect upon the several functions, but a mere fraction of this quantity given in half a minute may be sufficient to cause death.

Now, variations in the effects of chloroform in any particular animal appear, in ordinary circumstances, to be effected more powerfully by changes in the number and depth of the respirations than by any other factor. When chloroform is given by inhalation in the usual way (upon a cloth held near the mouth and nose), an amount of the drug which may be used with safety during ordinary easy breathing may speedily produce dangerous results when the respirations become rapid and deep. On some occasions I have tested this matter in a slightly different way—by causing an animal to breathe through the anæsthetic bottle, and observing the influence of certain definite doses of chloroform (*a*) during easy breathing and (*b*) during rapid gasping respiration, the state of the blood-pressure and the general condition of the animal being similar in both instances. The chloroform was given by being introduced into the anæsthetic bottle, through which the animal's (spontaneous) breathing was carried on. The difference in the effects of a few minims of chloroform in the two cases was very striking ; a dose that had only a slight influence during easy breathing was productive of powerful effects when given during rapid and deep respiration.

In experiments where artificial respiration was carried on by pumping air into the lungs, I have often tested the same point. Respiration was first carried on at a moderately slow rate (20 to 30 per minute), and the extent or depth of the respiratory movement was such as to resemble easy breathing. A certain amount of chloroform was injected into the anæsthetic bottle, and its effects on the blood-pressure, conjunctival reflex, etc., recorded. When the results of this administration had passed away, a change was made in the artificial respiration ; the movements were accelerated (to 60 per minute), and rendered more extensive; then chloroform in the same amount as before was introduced into the bottle. It was found that equal doses of the anæsthetic, administered in the two different conditions of (*a*) moderate respiration, and (*b*) exaggerated respiration, had remarkably different effects. With moderate respiration, the results of a certain dose (for example, two minims), were very slight, whereas the same dose during exaggerated respiration caused great depression—extensive fall of blood-pressure, etc. In these experiments the thorax was intact, and the animal was uninjured save for the introduction of a canula into the carotid artery for the purpose of obtaining a blood-pressure tracing, and a canula into the trachea for artificial respiration.

Similar results were obtained in experiments conducted with thorax laid open and recording apparatus applied to the heart, as described in Part II. of this Report. The minuteness of the doses of chloroform required to produce very marked results in many of my experiments (described in Part II.) was in close relation with the rapidity of the artificial respiration carried on. Such a respiration was necessary in order to avoid the manifestation of spontaneous efforts at breathing. Moreover, this rate is no greater than what is very frequently seen in the spontaneous breathing of the cat under chloroform.

From the great variation in the influence of a definite amount of chloroform, according to the rate and depth of the respiration, it is evident that the ensuring of free dilution of chloroform with air and the restriction of the amount of chloroform vapour to a certain percentage do not necessarily prove a safeguard against the speedy intake of an overdose. For air containing a sufficient percentage of chloroform vapour, for safe and effective anæsthesia during easy breathing, will, during deep and rapid respiration, supply a vastly greater amount of chloroform to the pulmonary capillaries. I am not able to state precise amounts for the volumes of air taken in during easy breathing and exaggerated breathing in the case of the cat. But we know that in man the quantity of air taken in at each respiration during easy breathing is about 20 cubic inches: whereas, during a very deep breath, about 100 cubic inches may be inspired. Thus five times as much air is taken in at a very deep breath as by an ordinary inspiration; but, in addition to the difference between ordinary breathing and deep breathing, we have to consider the results of acceleration.

In addition to a change in depth we have often a great change in rate. The rate may be doubled or trebled at the same time that the depth is increased. Hence it is evident that the amount of chloroform taken in during a period (for example, 10 seconds) of deep rapid breathing may be at least ten or fifteen times as much as during a similar period (10 seconds) of easy breathing. And, if there is a fixed percentage of chloroform vapour in the air all the time it is obvious that ten or fifteen times as much chloroform will be admitted into the lungs in the one period as in the other. Now, it will readily be understood that—especially if chloroform is already present and exerting its influence to some extent in the circulation—an overdose may be very speedily given in this way, and evidences of dangerous collapse may present themselves with striking suddenness.

The chloroform taken in will, of course, tend to lower the blood-pressure, and so tend to retard the access of the drug to the vital organs; but, as we have seen the heart often shows signs of dilatation before there has been sufficient time for the fall of blood-pressure to be brought about; hence in all probability in some cases an amount of chloroform sufficient to cause disastrous results may be taken in before a protective fall of pressure can come efficiently into play. Especially when deep rapid respiration is accompanied by a high or tolerably high blood-pressure, the amount of the anæsthetic rapidly taken in is apt to prove dangerous; and a fairly high pressure may be present for a time, even though a good deal of chloroform has been taken in. For example, when—although a considerable amount of the drug has been administered—the anæsthesia is not sufficiently complete, strong sensory irritation (for example, operative interference, or the introduction of too concentrated chloroform vapour into the air passages) can induce a marked rise of blood-pressure. The blood-pressure rises under the influence of sensory irritation, even though there is in the circulation an amount of chloroform sufficient to maintain a pronounced lowering of pressure in the absence of sensory stimulation.

Further disturbances of the breathing and the occurrence of deep rapid inspirations are apt to be excited by sensory excitation during imperfect anæsthesia, when the influence of the chloroform is not sufficiently great to prevent the respiratory centre being reflexly affected in this way. In this condition then there may be suddenly developed the conditions that are most favourable to the rapid intake of an overdose of chloroform, namely, quick gasping respiration, accompanied by a high blood-pressure.

At times sudden exaggeration of the respiration and a rise of blood-pressure may result from a temporary obstruction of the breathing. And in some instances it is possible that exaggerated respiration may be due to a directly stimulating effect of chloroform on the respiratory centre, but in this case it would probably not be so sudden in its development.

*Influence of certain Changes in the Action of the Heart and in the Blood-pressure upon the Effects of Chloroform Inhalation.*—I have seen no grounds for believing that changes in the action of the heart alone (for example, changes in rate) can influence the results of chloroform administration, except in so far as such changes affect the blood-pressure. A great increase in the rapidity of the heart's action may be attended by little or no rise in the blood-pressure. I have frequently seen a great and sudden acceleration of the heart without any important change of pressure ; and, conversely, a marked slowing of the cardiac rhythm may be accompanied by no fall of pressure. It is really the alteration brought about in the blood-pressure that is directly productive of marked effects in regard to the influence of chloroform. Changes in blood-pressure do undoubtedly modify in every important degree the results of chloroform or other inhalation. The great resistance towards both chloroform and ether during certain conditions of low arterial tension (for example, from vagus stimulation or section of splanchnics) has been already referred to, and is quite in accordance with the observations of the Hyderabad Commission on the same point. An amount of chloroform which may be given during a state of low arterial tension (depending on causes other than the anæsthetic) without marked effect may lead to grave results if the pressure be suddenly raised by artificial means, for example, by compression of the abdominal aorta. The dose of chloroform then reaching the vital organs is largely increased on account of the great augmentation of the blood stream in the head and upper part of the body. In this way a sudden change of pressure may be productive of dangerous effects.

During a condition of low arterial pressure the occurrence of an asphyxial condition may cause a great rise in the pressure, and thus lead to considerable variations in the amount of chloroform reaching the vital tissues, especially if the elevation of pressure be followed by the occurrence of exaggerated respiratory efforts, as may readily happen on the removal of a mechanical obstruction to breathing.

When a low arterial tension is dependent on the depressing influence of chloroform there is little or no chance of any important rise of pressure being brought about by irritation of sensory nerves ; the anæsthetic prevents the occurrence of such a vascular constriction from sensory excitation.

But during *imperfect* anæsthesia a moderate pressure may suddenly be converted into a high pressure by strong irritation of sensory nerves, causing stimulation of the vaso motor centre and increased vascular tone. When such an elevated pressure is combined with violent respiratory efforts, there is danger of an overdose of chloroform being taken in unless great care be exercised.

The danger involved in the abrupt change from a low pressure to a high pressure is due to the fact that when the pressure is low chloroform may be given in comparatively large amount without producing any striking effect ; the chloroform may be pushed, and then when the pressure suddenly rises dangerous effects may speedily ensue if the administration of the anæsthetic is continued in the same way (in the same strength) as was employed during the state of low pressure ; the amount of chloroform reaching the vital organs becomes much augmented with a high pressure. It is evident, then, that a low blood-pressure must last for some time, so as to lead to a more abundant administration of chloroform, before any danger can, with reasonable care, be caused by a change to a higher pressure. Hence the occurrence of a very brief and transient phase of low pressure (such as may be caused by a short inhibition of the heart), followed by a speedy rise to the normal height, does not introduce any special danger in the use of chloroform ; for the period of low pressure does not last long enough to induce the administrator to give chloroform more abundantly, and thus there is no danger in the subsequent rise.

Apart from the influence of asphyxial conditions, a change from a *very low* pressure to a *high* pressure is very rare under chloroform. Such a change can, of course, be easily brought intentionally (for example, by stimulation of the vagus, compression of the vena cava inferior, *etc.*), and it may possibly occur in certain peculiar circumstances of operative procedure, but its occurrence in such a degree as to constitute a serious danger in chloroform administration is probably rare.

On the other hand, it has been already stated that during imperfect anæsthesia a sudden change from a *moderate* to a *high* pressure (caused by sensory irritation) may co-operate with exaggerated respiration in leading to the intake of a dangerous overdose.

*Indications of Danger during Chloroform Administration derivable from the Respiration and from the Blood-pressure.*—The relative importance of observation of the respiration and the blood-pressure with regard to the warnings of danger derivable from them was studied in many experiments in the following manner : A cat, rendered unconscious with chloroform in the usual way, was fastened on a Czermak's holder ; a blood-pressure tracing was taken (by means of a Ludwig's kymograph) from the carotid artery, and a tracing of the respiration by a tambour kept in contact with the animal's chest and communicating with a recording tambour which inscribed its movement on the recording surface simultaneously with the blood-pressure tracing. More chloroform was then given on a cloth held near the animal's mouth and nose, and after a time the anæsthetic was pushed. The course of the blood-pressure and the respiration was accurately registered by their tracings ; it was found that indications of approaching danger were given most commonly by the respiration, but this was by no means *invariably* the case. In several instances warning was given first by the blood-pressure. A sudden and precipitous fall of pressure occurred at varying periods—fifteen, twenty-five, thirty seconds, and even longer—before there was anything in the state of the respiration to cause alarm, and even then the respiration did not stop, but became slowed and somewhat spasmodic in its character. Sometimes a slight change either in rate or force is seen in the respiratory tracing along with, or very soon after, the fall of pressure, but such a change would not have been apparent to the eye without the use of accurate recording apparatus ; and even when visible to the eye, the respiratory changes accompanying a dangerous fall of pressure were sometimes not more marked or notable than alterations in the respiration occurring at earlier phases of chloroform administration and attended by no serious consequences, though the giving of the anæsthetic was not suspended, or any remedial measures adopted.

The sudden and precipitous fall of blood-pressure above referred to as giving warning of an overdose was, I have no doubt, attended by a marked change in the pulse—such a change as could have been appreciated had the pulse been felt during its occurrence.

The period intervening between the indication of danger given by the blood-pressure and that afforded by the respiration in these cases is a short one, but there can be no doubt that in such cases the gaining of even a very short time may be of extreme importance. The difference of a very brief period as regards the moment when the further administration of chloroform is stopped, and remedial measures adopted, may turn the scale in favour of recovery or death.

## PART V.

### *General Conclusions.*

1. During chloroform anæsthesia the blood-pressure is lowered and the heart's action is weakened.

2. Dilatation of the heart occurs to an appreciable extent, even when chloroform is administered gently, mixed with abundance of air (under 4 per cent. of chloroform vapour in the air).

3. Dilatation may occur even before the conjunctival reflex is abolished.

4. The dilatation affects all parts of the heart more or less—the left side as well as the right. It is not due to changes in the pulmonary circuit.

5. The dilatation is not due to the accompanying fall of pressure, to the diminished resistance to the ventricular systole, or to the diminished blood-supply through the coronary arteries. Dilatation does not result from a similar fall of pressure brought about by means other than chloroform, for example, arterial relaxation caused by section of vaso motor nerves. Dilatation under chloroform often occurs very quickly, before there is any fall of pressure. Moreover, when the dilatation has followed a fall of pressure it is not removed by artificially raising the pressure, for example, by compression of the abdominal aorta.

6. There is no distinct change in the rate of the heart's action when dilatation occurs. A sudden and complete cessation of the cardiac rhythm is never caused by the inhalation of chloroform. Cardiac failure occurs by a more or less sudden enfeeblement and dilatation of the organ ; not by a sudden complete cessation of rhythm.

7. The tone of the heart muscle is depressed, the cardiac walls become relaxed, and the functional efficiency of the organ is impaired.

8. When the heart becomes greatly dilated it fails to be an effective force in keeping up the circulation, while its rhythmic movement still continues—though so feebly as to be inefficient.

9. Cardiac failure sometimes occurs in this way a considerable time before the respiration stops, though generally the respiration stops before the heart has become incapacitated.

10. The failure of artificial respiration to bring about recovery (in some cases of chloroform collapse), when begun immediately after the spontaneous respiration has ceased, is in all probability due mainly to the enfeebled and distended state of the heart, which has become unable to maintain the circulation. Hence the supply of fresh air (by artificial respiration), free from chloroform, cannot be taken advantage of.

11. The depressing influence of chloroform on the heart—leading to dilatation of its cavities—is not exerted through the vagus nerves, but is a direct effect of the drug upon the cardiac mechanism. Section of both vagi does not obviate the weakening and dilating influence of chloroform upon the heart.

12. The weakening and dilating effects of chloroform are sometimes manifested in tolerably equal degree on both auricles and ventricles ; but sometimes more readily upon the auricles, and at other times upon the ventricles.

13. A peculiar periodic depression of the ventricular action sometimes occurs during recovery from the primary effects of chloroform.

14. The contrast between the relation to the heart's action of chloroform and ether in anæsthetic doses is very marked. With chloroform, cardiac dilatation frequently occurs—and often, indeed, a very marked dilatation—before the conjunctival reflex is abolished. With ether, the induction of anæsthesia with complete abolition of the conjunctival reflex has not been attended by any noteworthy dilatation ; indeed effects of a stimulating character have sometimes been observed, and the peculiar periodic ventricular depression sometimes following chloroform has been seen to be removed.

15. Under the influence of chloroform a temporary slowing of the heart's action sometimes occurs—from asphyxial conditions or from sensory stimulation during imperfect anæsthesia. This slowing is quite different in its nature and causation from the enfeebling and dilating effect already mentioned. The slowing is not due to direct influence of chloroform on the heart, but is indirectly brought about through the vagus nerves. It does not appear to be dangerous in the healthy animal.

16. The occurrence of fibrillar contraction (delirium cordis) does not appear to be a primary mode of cardiac failure from the inhalation of chloroform in the healthy animal, though it may sometimes supervene when the heart has become distended and incapacitated by chloroform.

17. The fall of blood-pressure under chloroform is in its earlier stages due mainly to the depressing effect of the anæsthetic on the vaso motor centre, preceded often by a slight stimulation ; the later stages are associated with failure of the heart as well as of the vaso motor centre.

18. The relative occurrence of cardiac dilatation and vaso motor depression varies. Sometimes the heart begins to dilate early—before there is any fall of pressure ; at other times a large fall of pressure may occur before cardiac dilatation becomes marked.

19. The lowering of the blood-pressure is in a certain sense protective ; it retards the access of more chloroform to the vital organs. But, on the other hand, the fall of pressure may become excessive and produce dangerous effects.

20. In certain circumstances, when chloroform is very suddenly taken in, a dangerous dose may be absorbed, and the heart may become seriously affected *before* the vaso motor centre has had time to be much depressed.

21. When a fall of carotid pressure has been brought about by the *gradual* inhalation of chloroform in the ordinary way, firm pressure applied to the abdomen causes a marked rise of pressure—very much more than can be obtained by inversion of the animal. And even when the fall of pressure is due to the *sudden* inhalation of an excess of chloroform, pressure on the abdomen commonly, but not in all cases, leads to a decided rise in the carotid pressure. The existence of cardiac failure may prevent the possibility of such a change.

22. Changes in the respiration exert a most important influence upon the effects of chloroform administration. An amount of chloroform which can be given with safety during easy breathing may speedily become dangerous during deep, rapid respiration.

23. Free dilution of chloroform with air—the restriction of the percentage of chloroform vapour to 4 or 4½ per cent.—gives no security against an overdose. A percentage that gives safe anæsthesia during ordinary breathing may lead to fatal collapse if given during exaggerated respiration.

24. Changes in respiration may be excited by sensory stimulation (operative interference, too strong chloroform vapour, *etc.*,) during imperfect anæsthesia. Rapid, gasping respiration occurring in such circumstances is usually accompanied by a rise in the blood pressure, and, as there may be already a considerable amount of chloroform in the circulation, there occurs a combination of circumstances specially favourable for the speedy and sudden development of dangerous collapse.

*Note.*—I hope soon to publish tracings illustrative of some of the experimental facts described in this report.

# ARTICLE, REPRINTED FROM *THE MEDICAL CHRONICLE* OF APRIL 1890.

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THE opinion that, in a certain number of cases, death from chloroform is due to cardiac paralysis, was first enunciated by Dr. Sibson (*London Medical Gazette*, Vol. XLII., p. 109), and it is important to note that this conclusion was originally arrived at, not from physiological experiment, but from a consideration of the first fatal cases in man.

There appears to have always been some contradiction between the results obtained by experiment on animals and those obtained by clinical experience on human subjects. Thus, as early as 1848, Mr. Thomas Wakley proved, by a series of experiments on animals, that in deaths from chloroform the heart continued to beat after cessation of the respiration, these results being confirmed by a Commission held in Paris in 1855 (*L'Union Medicale*, No. 13, 1855).

Notwithstanding these experiments, the reported fatal cases from chloroform inhalation appeared to indicate that in man, at least, the failure of the heart preceded the cessation of respiration. The grounds for this belief are that the fatal result is usually sudden, the patient in the midst of violent struggles, after a deep inspiration, becoming at once practically lifeless, the pulse stopping before the respirations, and occasionally becoming distinctly and gradually feebler before cessation of the respirations. But, no doubt, the main reasons for the belief were the suddenness of the result, the cessation of the pulse, and the non-success of artificial respiration to restore the patient.

Dr. Snow was the first to reconcile the difference between the results of experiment and of clinical practice, by showing that death might happen in one of several ways, according to the amount of chloroform absorbed—

(*a*) Chloroform inhaled gradually, until death resulted, killed by gradual paralysis of respiration, the heart continuing to beat after cessation of the respiration.

(*b*) But when a large dose of the drug was suddenly taken into the system, the heart ceased beating before the respiration stopped.

(*c*) It was also thought that this sudden paralysis might set in before insensibility was produced. The cardiac paralysis was considered to be the chief factor in the fatal result, because efficient artificial respiration did not resuscitate these cases.

There was an impression that the cessation of the respiration might be entirely remedied by artificial respiration, but that the affection of the heart was necessarily fatal.

Clover, writing about 1875, says of chloroform : " . . . besides its liability to cause apnœa, which is remediable, it may produce total arrest of the heart's action, which is not so."

These views of the action of chloroform were confirmed by the Commission of the Medico-Chirurgical Society, London, in 1864, and again in 1879 by a committee of the British Medical Association, in their report on the action of anæsthetics (*British Medical Journal*, 1879, Vol. I., pp. 1 and 921), stated that " chloroform has sometimes an unexpected and apparently capricious effect on the heart's action. Chloroform may cause death in dogs by primarily paralysing either the heart or the respiration. The variations in this respect seem to depend to some extent on individual peculiarities of the animals (?). In most cases respiration stops before the heart's action, but there was one instance in which respiration continued when the heart had stopped, and only failed a considerable number of seconds after the heart had resumed."

The whole work of the committee tended to prove the conclusions arrived at by Dr. Snow, that under certain conditions chloroform did affect the heart before the respiration, though failure of the respiration was the most common cause of death.

The Hyderabad Chloroform Commission have come to a conclusion similar to that arrived at by Mr. Wakley in 1848, and the Commission of the Society of Emulation of Paris, in 1852, *viz.*, in death from chloroform the heart continues to beat after the respiration has ceased, results in contradiction to the previous London Commission of 1864, and the Glasgow Commission of 1874.

There may be several objections taken to the unreserved application of the conclusions of these experiments to man.

First, there is the difference which may exist in the action of chloroform on the heart of man and of animals. There is the possibility that the habits of life, the taking of stimulants and narcotics, especially the habitual use of drugs like tobacco—for the condition of the vagus centre is shown to be of great importance in relation to chloroform narcosis—may have some effect, if not in altering the behaviour of the heart muscle or its innervation, at least in modifying that chain of events upon which depends the absorption and distribution of the chloroform by the blood. Amongst other points of difference between man and the animals experimented on are the relation of the vital capacity to the size of the animal, and the interference with complete filling of its lungs, resulting from holding of the animal.

It has often been shown that the suddenly fatal cases are rare in human subjects in which there is any impediment to the free expansion of the chest, *e.g.*, Clover has pointed out that a phthisical patient is less likely to take a fatal dose of chloroform than one with healthy lungs, because his chloroform-absorbing capacity is diminished. Again, it has been noted that few patients die suddenly from chloroform, when it is inhaled lying on the side; also few, if any, sudden deaths are reported in case of ovarian tumours. The explanation of these cases is that the interference with full expansion of the lungs by the position or the presence of the tumour prevents the lungs taking in the necessary fatal dose.

On the other hand, the greater number of the suddenly fatal cases occur in people with healthy chests and large vital capacity, *e.g.*, in reducing dislocations of the shoulder, and in minor operations; such as teeth extraction, especially in those in which the patient, being in the natural upright position, can give the respiratory muscles full play, and completely fill the chest.

Thus it is worth considering if the holding of a struggling animal, such as a dog, might not so interfere with its breathing as to prevent the full dose being taken, especially as the animal would not be likely to be held in its natural position. There is an indication in the report that, in at least some cases, the holding of the animal did actually interfere with its respiration. If it did so in one case it probably did so in many, especially as a dog's thorax is so shaped, that to hold its fore legs together in front of it, would prevent the complete expansion of its chest, and so prevent it suddenly taking a fair dose of chloroform vapour.

In animals, too, we have an absence of the desire to inhale freely. In at least one reported case the wish to inhale the chloroform freely contributed largely to the fatal result,

In chloroform accidents in man, of the very sudden variety, considered to be due to cardiac syncope, a certain set of conditions are present which are difficult to obtain in animals.

The subject is generally young or middle aged, with an expansile chest ; the chloroform is willingly inhaled, quietly at first, until semi-unconsciousness is produced, when the fauces and glottis are insensitive ; then, during struggling, with or without holding the breath, the patient, often in an upright or semi-upright position, and having his arms fixed by the assistants, which gives his respiratory muscles good purchase, gets one or two deep inspirations at the greatest advantage, and so obtains the maximum amount of chloroform.

There is the desire to get under the influence of the drug the voluntary inhalation followed by the still steady conscious anxiety to inhale more, and then the involuntary deep inspiration, in the natural position in which a deep inspiration can be best taken.

All these are conditions difficult to obtain in experiments on animals, and it is only under such conditions that the supposed cardiac paralysis has been produced in man.

The interval between the stoppage of the heart and the respiration is so short that these points, trivial though they seem, may easily be of importance in modifying the result.

It has always been maintained by the advocates of the syncope theory that it is only under certain circumstances, and by a certain definite combination of factors, that chloroform sufficient in quantity to paralyse the heart can be introduced into the blood, and that this particular combination of circumstances occurs very seldom—about once in every 1,000 or 2,000 administrations.

Thus as Dr. Snow pointed out years ago, the effect of chloroform on any given case varies with the amount contained in the blood at any given time.

This depends upon the strength of chloroform vapour exhibited to the patient, the ease with which it is inspired through the glottis, and the depth and frequency of the inspirations ; the amount actually absorbed depends upon the condition of the blood (its capacity for chloroform may vary), the time it stays in the lungs, the rapidity of the circulation, and other factors.

The idea was that by certain combination of these several factors, the maximum quantity of chloroform was absorbed by the blood in the lungs and hurried to the heart, the next organ in the order of the circulation, not merely distending its cavities with chloroform-saturated blood, but also being distributed by the coronary arteries to every part of its substance, and so causing paralysis. This result is also assisted by the condition of distension of the heart and the venous condition of the blood.

That the arrangement of these various factors—respiration, pulse, *etc.*,—has an important bearing in the action of the drug on the heart, is well shown by these experiments of the Commission. These showed that by varying the methods of administration in different experiments it was possible to make the heart cease beating at intervals of from one to twelve minutes after the cessation of the respiration. It is not impossible that by further variations the time might have been reduced until the stoppage was simultaneous with, or even preceded the cessation of respiration, and the experiments would have been thus made to accord with certain reported fatal cases in man.

The cases in which the heart ceased soonest after the respiration were cases complicated with asphyxia, that is, cases in which semi-asphyxia was produced, presumably followed by deep inspiration ; under these circumstances the heart ceased beating one minute after the respiration stopped. Now it is under conditions similar to these in man, that the so-called cardiac syncope occurs, and it is by working on these lines that attempts to produce it in animals should be made ; but it appears that the Commission only made some four of these experiments hardly a sufficient number upon which to base an important statement.

In ordinary practice, the several conditions giving a fatal result from cardiac syncope are combined about once in every two or three thousand administrations. Because the Commission did not obtain it in some 600 attempts they conclude that it cannot exist. This is scarcely reasonable, especially as the bulk of their work was not upon the lines which promised the nearest result, *viz.*, those of deep inspiration after semi-asphyxia.

The manner or means by which the movement of the heart was tested are of some moment, as explaining the discrepancy between the results of experiment and practice. Most of the cases were tested by a needle introduced into the heart, or by opening the chest. These methods will indicate the merest quiver of the heart muscle,—movements that would give no indication of their presence to ordinary observation, and movements that would probably be of no functional use, *i.e.*, in no sense contractions. It has been noticed by other observers, that the hearts of animals killed by chloroform show fibrillary irritability, after the heart muscle, as a whole, is incapable of contraction,—a condition similar to this would indicate movement tested by the above, but such movement would be fallacious.

It would be interesting to know the degree of action exhibited by the heart after cessation of the respiration. Was the power of propelling blood retained ? If the action consisted only of muscular tremors it would be of little value, and hardly what is understood by action of the heart.

The following case may be here quoted as a good example of the class of chloroform accidents, upon which is based the theory of the action of chloroform on the heart :—

The patient was a strong, healthy labourer, who required an anæsthetic, while some adhesions were broken down in his knee-joints ; he had taken chloroform successfully three months previously. The case is reported by the surgeon who was superintending the administration of the anæsthetic. Chloroform was administered by a towel. (*British Medical Journal*, 1884, Vol. II., p. 811.)

"The patient showed not the least sign of nervousness . . . and commenced inhaling by several rapid shallow breaths, as if determined to get quickly under the influence of the anæsthetic. Following immediately upon the shallow movements were three or four deep inspirations. Feeling that this method must have entailed a large and rapid consumption of chloroform, I directed the house surgeon to remove the cloth, when the patient, *though still breathing*, was seen to have his eyes wide open, the pupils rapidly dilating, and the conjunctiva insensible. The face was neither livid nor pale ; the lips, indeed, were of their normal pink hue, and the cheeks slightly flushed. In fact, beyond the condition of the eyes, there was nothing in the facial aspect to excite alarm. Up to this point, the time of the inhalation could not have exceeded one to two minutes. On feeling the right radial no pulse could be detected. The head was lowered, respiration then ceased ; not, however, until three or four breaths had been drawn after the recognised cessation of the pulse. After the commencement of artificial respiration, the patient spontaneously made several gasps, but there were no futher signs of vitality."

This case is valuable because it is reported, not by the administrator of the anæsthetic, but by one who was merely watching the case, and who had ample time accurately to note the events which occurred ; and the absence of struggling, so common in suddenly fatal cases, made the observation of the symptoms easy.

Taken in conjunction with the numerous reported cases in which the patient passes suddenly from a condition of active struggling to a state of collapse, with gasping respiration and absence of pulse, it proves that chloroform, if it does not actually paralyse the heart, may cause death by some means equally sudden and certain ; and that in such cases, the heart is practically stopped, as far as its functional value is concerned, before the respiration. The case indicates also that observation of the respiration alone will not give adequate warning of danger, for the respiratory movements may be perfect one second, and the next be on the verge of extinction, and hopelessly beyond recovery. It also shows that fatal damage may have been wrought on the system before there is any outward sign.

This case is also noteworthy as contradicting the statements made by the first Hyderabad Chloroform Commission that since 1856, no death from chloroform has occurred in which the respiration, and the respiration alone, was attended to throughout the administration.

The patient was breathing perfectly, and there was no sign of respiratory failure when the administration was suspended, so that was the respiration alone observed it would only have signalled any danger some seconds later. There can be little doubt but that the pulse in such a case, where the patient was quiet and breathing tranquilly, would have exhibited some decided alteration had it been observed.

In the second portion of the report it is shown that the essential action of chloroform on the system is to cause a fall in the mean blood-pressure. This fall is proportionate to the amount of chloroform inhaled and absorbed into the system. [It would have been interesting to have known the relation, if any, the condition of the blood-pressure bore to the state of anæsthesia.]

This fall in blood-pressure bears some relation to the respiration, but what this relation is, is not distinctly stated, and the cause of the fall in pressure is also not clearly assigned, except (Sec. 19) that it is not due to the direct action of chloroform on the heart, and must be due to paralysis of the vaso motor centre. This is important when taken in conjunction with the following parts of the report (Sec. 3) : " . . . If the chloroform is pushed further, there comes a point not easy to define when the blood-pressure and respiration will no longer be restored spontaneously, although the heart continues to beat after the inhalation has stopped." And also (Sec. 25) : ". . . It is never in any case certain that artificial respiration will restore the natural respiration and blood-pressure, *no matter how soon it is commenced* after the respiration stops."

This exposes a comparatively new and insidious danger in chloroform administration, and one as difficult to treat as the old cardiac syncope. It is quite conceivable that under certain circumstances the dose necessary irretrievably to damage the vaso motor centre may be inhaled with extreme rapidity, and that while the respiration and pulse are still present, the centre may be almost paralysed before much warning has been given.

From a record of fatal cases in man, it appears possible that this paralysis may, in certain conditions, be effected almost instantaneously, and before complete insensibility has been produced.

In Section 20 injection of chloroform into the jugular vein caused no paralysis of the heart. It should be noted that this is not the most direct channel by which the heart can be reached ; and the experiment, when compared with inhalation, is open to criticism. Chloroform injected into the jugular vein passes with the venous blood to the right auricle, where it is mixed with the blood of the inferior vena cava from the liver ; from the right side of the heart it passes to the lungs, where the chloroform, being volatile, and the air in the alveoli of the lungs free from chloroform, a good deal may be lost ; finally, what remains of it reaches the heart.

Chloroform given by inhalation is much more likely to reach the heart directly in large doses passing into the air cells ; about one-fifth of the whole blood of the body is at once exposed to it. This blood, saturated with chloroform and unmixed with any non-chloroformed blood, goes direct into the left auricle and ventricle, and by the coronary arteries is first distributed into the very substance of the heart itself. As Dr. Snow puts it : " In inhalation the heart is always a little in advance of the rest of the body as regards the amount of chloroform vapour to which it is subjected." Chloroform thus given by inhalation may have, a more potent action than when injected into a vein, and the results of the two methods cannot well be compared.

In Section 12 the effect described as that of asphyxia on the blood-pressure, in causing a rapid fall of blood-pressure and slowing of the heart beat, is contrary to the received ideas on the subject. (" Human Physiology." Landois and Stirling. 1888 edition, p. 198.) During asphyxia, " the blood-pressure measured in a systemic artery, *e.g.*, the carotid, rises very rapidly, and to a great extent during the first and second stages ; the pulse beats are at first quicker, but soon become slower and more vigorous. The rise in pressure is due to stimulation of the general vaso motor centre by the venous blood, and the slow beating of the heart to the action of the venous blood, on the cardio inhibitory centre in the medulla."

The attempts to produce shock during the inhalation of chloroform (§ 33) uniformly failed. In experimenting on animals there are several sources of error. In the first instance, it is doubtful if animals suffer as much from shock as man, there is an absence of mental terror and the depressing effect of the anticipation of the operation, &c.

It is well known that the extraction of a single tooth in a state of semi-anæsthesia may produce a considerable amount of shock and a painful mental impression that lasts for weeks,—an amount of shock far greater than would have been felt had no anæsthetic been exhibited.

The lowering of the blood-pressure in the case of a fatty heart (§ 39) cannot be altogether an advantage in chloroform administration. It must, by the efforts the heart makes to keep up the pressure, increase the work of the heart, and at the same time diminish the blood supply to the heart itself.

It is important to note that in all these experiments there are double and treble reactions, which, though they have a beneficial effect in diminishing the amount of chloroform absorbed, are in themselves elements of danger.

Thus, semi-asphyxia and inhibition of the heart are elements of safety by preventing the " intake " and distribution of chloroform, but the ultimate result of this is to cause rapid breathing and quick pulse, which, by the extra " intake " of chloroform and the quick distribution of that already inhaled, is an element of danger.

This change from a slow to a rapid pulse may be attended with very nearly fatal results, as is shown in Section 34, where an animal was nearly killed by a short inhalation, from the inhibition of the vagus having been stopped, and a more rapid pulse consequently resulting. This fall in pressure is in itself, in feebly nourished subjects, a source of danger, causing syncope from cerebral anæmia.

There is a peculiar animus against the feeling of the pulse displayed by Surgeon-Major Lawrie in the first Report, and not entirely absent in the second. He goes so far as to state, " that since the year 1855, in Great Britain, there is no death from chloroform recorded in which it was proved that the respiration alone was attended to throughout the inhalation." This is surely a mistake ; there are several fatal cases where the respiration only was attended to, one of which is quoted here, and there are many more in which respiration was attended to as fully as possible.

Then reference is made to the teaching of what are termed antagonistic schools : Syme taught—" We are guided as to the effect of the chloroform not by the circulation, but entirely by the respiration." Erichsen teaches : " When fully anæsthetised the patient requires the most careful watching by the person who administers the chloroform ; his finger should never be off the pulse, nor his eye taken away from the countenance of the patient."

Surgeon Lawrie considers these opinions "absolutely irreconcilable". They are nothing of the kind. Erichsen's directions are only a little fuller and more complete than Syme's, and not incompatible with his.

I do not know the wording of the earlier editions of Erichsen, but in recent ones (edition of 1884, Vol. I., p. 24), in addition to the words quoted, it is stated, " The breathing must be carefully observed." It is unlikely that it is not mentioned in some part of the earlier editions, especially as Dr. Snow, with whose works Mr. Erichsen would no doubt be familiar when writing the above, constantly emphasises the necessity for observing the state of the respiration and in reference to this point says :—" The importance of attending to the respiration of the patient has previously been noticed, and it is so obvious a symptom that it can hardly be disregarded if any one is watching the patient ; it speaks, moreover, almost to one's instincts, as well as to one's medical knowledge." (" Snow on Anæsthetics," p. 258, 1888).

Apart from any action which chloroform may have on the heart itself, the experiments made supply abundant reason for a careful observance of the pulse during the administration of the drug, and it is surprising that this point has not been referred to in the Practical Conclusions. Its omission looks suspiciously like the result of prejudice against it.

In the report it is shown that the amount of chloroform inhaled (the " intake ") depends upon the respiration, but that the effect of this " intake," the way in which it is utilised, lies with the circulation, with the force and rapidity of the pulse or the reverse.

It is shown that, with a slow pulse, concentrated chloroform vapour can be exhibited with impunity, but with a full, bounding pulse dilute chloroform vapour may be dangerous. These variations occur in practice, from the quick full pulse of a patient with a high temperature to the slow pulse of a patient with jaundice.

There have been fatal cases in man distinctly due to a non-consideration of these points. [Case reported in *British Medical Journal*, 1887, Vol. II., p. 951, where ether was given first, and then a small dose of chloroform proved fatal.] This is again illustrated in Experiment 177, Section 34, where sudden quickening of the pulse, from cessation of vagus inhibition, suddenly placed the animal in a condition of " extreme and unexpected jeopardy". Yet from some reason, though stress is laid upon these points in the Report, they are omitted from the Practical

Conclusions. This is the more strange, as the Commission certainly proves that the circulatory apparatus is first influenced by the chloroform, and it is by vaso motor paralysis that death is caused. Why, then, disregard it in administering the drug?

As attention to the pulse is so easily managed without neglect of the respiration, and a proper knowledge of the state of the circulation has been proved to be most essential to the intelligent administration of an anæsthetic, it is advisable that the pulse should always be observed; it can, at least, do no harm, and, in conjunction with the respiration, is a valuable help, quite apart from any information it gives of the general condition of the patient.

The assistant in charge of the anæsthetic must endeavour to be more than a mere doler out of innocuous doses of chloroform; he should keep himself in touch with the whole condition of the patient all through the operation.* To endeavour to narrow his work to mere watching of the respiration is mistaken policy.

In reference to the question of cardiac *versus* respiratory paralysis, the following remarks appeared in the *Lancet* for September 21, 1889.

"In the Scotch capital, failure of respiration is regarded as the chief or only danger; while, in the metropolis, failure of the heart is more feared. It is quite possible that the surgeons in both cities are right, and that the habits or mode of living of the people may lead to differences in resisting power of the cardiac or respiratory apparatus respectively. The proportion of gouty patients is much larger in London than in Edinburgh, and when we consider that the natives of India appear to resemble the Scotch in their comparative immunity from cardiac paralysis by chloroform." . . . . As most cases of death from chloroform are put down as due to cardiac paralysis, and this is said to be rare in Scotland, the impression is conveyed that fatal cases of chloroform narcosis are unknown or rare in that country, and there is certainly an idea abroad to this effect. It is not necessary to resort to a comparison of the habits of the people to explain their immunity from fatal results.

In Scotland fewer people die from chloroform, simply because fewer people inhale it. A population of 3,991,499 must necessarily give fewer chances for the administration of chloroform than one of 28,247,151 and consequently fewer possibility of fatal cases. Another point is, that such cases as do occur are not always, from the absence of coroners' inquests, brought to light a large number, if not the majority of the fatal chloroform cases in England, owing their publicity to the newspaper reports of the coroners' inquests.

But deaths from chloroform *do* occur in Scotland and even in Edinburgh. A casual search in the medical journals gives the following figures: Edinburgh Royal Infirmary (1878 to 1880), three deaths; Western (Sir George Macleod's Hospital) Infirmary, Glasgow (1883 to 1885), three deaths; Royal Infirmary, Glasgow (1883 to 1885), two deaths; other parts of Scotland (1881 to 1883), six deaths. England, 110 deaths; Scotland, 14 in same period. England, population 28,247,151; Scotland 3,991,499.

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[* *Vide* Note by the Commission at the end of Mr. Wilson's Article.]

In summing up the results of the latest Commission on chloroform it is to be remembered that, in the opinion of many persons, as regards the first part of their work, *viz.*, the investigation of the point, does chloroform paralyse the heart? nothing new has been added to our knowledge.

The result has been to confirm the work of some of the previous experimenters, but without explaining how the theory of cardiac paralysis appeared to be borne out by the experiments of Dr. Snow. There has always been since the discovery of chloroform sufficient evidence to prove that it is very difficult, if not impossible, to produce sudden cardiac paralysis by it in animals, and an equal amount of clinical evidence to justify the supposition that such can occur in man.

To estimate the value and importance of the second part of the report, it must be noticed that the action of chloroform on the heart was (by the advocates of this anæsthetic) considered to be the *only* objection to its general use. It was this supposed power of causing irremediable cardiac paralysis which made people afraid of it. If this property could be shown to be non-existent the harmlessness of the drug would be established.

This is easily understood, from the facts that artificial respiration is so readily carried out, and cessation of respiration remedied by artificial respiration so common, that the impression is prevalent that it is easy to breathe for a person, but the remedying of the cessation of the heart-beat presents insuperable difficulties.

It was not imagined that chloroform might possess other dangers—it is for a knowledge of these that we are indebted to the Commission, *viz.*, the importance of the part played by vaso motor paralysis in the causation of death, and the fact that the paralysis of the respiratory centre may be final at the moment that attention is drawn to the cessation of respiration.

If the conclusions of the Commission are accepted in full without reserve, chloroform has no power of paralysing the heart; one danger is removed, but it is replaced by one as great in every respect, *viz.*, paralysis of the vaso motor centre. This may set in very suddenly, with hardly any warning; it is as far beyond treatment as cardiac paralysis, and it is as fatal. If the cardiac paralysis from chloroform does not exist, vaso motor paralysis must have been the cause of the numerous deaths recorded against chloroform; and how sudden and irremediable this is the reports of fatal cases abundantly testify.

When death occurs from vaso motor paralysis it is clearly imaginable that the vaso motor centre may be hopelessly damaged or rapidly reaching that stage, while the pulse is still present and the heart attempting to keep up the pressure. So that for some seconds there may be a deceptive condition in which both pulse and respiration are present, yet a fatal termination is imminent. That this death from vaso motor paralysis does occur in man is shown by the very rapid running pulse mentioned in some cases as preceding the fatal termination.

The extensive experiments of the Commission have left the chloroform question in the following condition: It was not found possible to directly paralyse or affect the heart by chloroform in some 600 administrations. Death from chloroform is due apparently to paralysis of the vaso motor and respiratory centres—probably one or both of these may be affected. When death occurs it is the result of an overdose of the drug.

It cannot be too strongly insisted that the work of the Commission gives us no greater confidence in chloroform than we had before. That it does not cause cardiac paralysis is no safeguard, since it produces as sudden and equally fatal vaso motor paralysis, and respiratory paralysis in addition. Its physical and chemical properties remaining the same, the danger of permitting the patient to inhale an overdose will remain as great as ever.

In the Practical Conclusions no directions are given for its administration beyond those which have been in use for years, and in the presence of which so many fatalities have occurred, either from vaso motor, cardiac or respiratory paralysis.

In the face of these it is hardly to be expected that the deaths from chloroform will much diminish.

[The Hyderabad Commission has proved that the sole duty of the Chloroformist is to produce normal anæsthesia, and we agree with Mr. Alexander Wilson that this duty includes keeping "himself in touch with the whole condition of the patient all through the operation". But keeping in touch with the patient's condition is a very different thing from taking the pulse as a guide to the effect of chloroform, for the simple reason that the pulse affords no indications whatever of normal anæsthesia. The pulse is dangerously affected by abnormal chloroform administration; and abnormal inhalation alone can originate the deadly symptoms of peril for which Mr. Wilson watches it.]

## ARTICLE BY SURGEON-MAJOR E. LAWRIE.

[*Reprinted from the "Medical Chronicle" of January* 1891.]

IN the MEDICAL CHRONICLE of February, 1890, there is an ably written review of the Hyderabad Commission's report on "Chloroform," signed "Alexander Wilson." The review ends thus:—"Increased knowledge has added nothing new to the directions for its" [chloroform's] "administration." I hope that on reconsideration Mr. Wilson will see fit to modify this statement, which is inconsistent with fact.

The Hyderabad Commission was appointed to confirm or disprove Syme's and Simpson's principles that we should be guided as to the effect of chloroform entirely by the respiration. The Commission has not only proved that these principles are sound, but has also proved that the art of administering chloroform with safety consists in keeping the breathing absolutely regular throughout the inhalation. This proof is new, and has never been established before. As far as I am aware there is no text-book on surgery of the present day, except Sir Joseph Lister's article in "Holmes's Surgery," that does not recommend that the pulse as well as the respiration should be watched for signs of the effect of chloroform, and it is new to prove, as the Hyderabad Commissior has proved, that to watch the pulse at all is both wrong and dangerous.

The photograph of two original tracings of Experiment No. 186 which accompanies this paper demonstrates the futility of attention to the pulse in chloroform administration. For example, in Tracing III., Ludwig, though the pulse of the dog experimented on was apparently much better at 2 hours 55 minutes than at 2 hours 54 minutes, it was not really so. Dr. Gaskell has shown that when the blood-pressure is high the pulse must necessarily be better than when the blood-pressure is low. The pressure at 2 hours 54 minutes was much higher than at 2 hours 55 minutes, and the pulse was therefore in reality better in proportion, though it was smaller in the tracing.* On the other hand, the danger of waiting for signs in the pulse of failure of the heart under chloroform is well shown in the Tracing IV., Ludwig, at the end of which the failure of the pulse at 3 hours 19 minutes 15 seconds, was a sign of impending death.

The two tracings demonstrate all that it is possible to know regarding the safe administration of chloroform, and are well worth careful examination. (Everything which occurred in the experiments was marked and recorded on the tracings as the drum revolved, and nothing except what is written in red ink† was added afterwards.) The most remarkable points they exhibit are (*a*) the uniformly gradual and regular fall of blood-pressure which occurs when chloroform is administered in such a way that the breathing is perfectly regular, and (*b*) the entire absence of danger if the administration is stopped when the animal or patient is "under". Formerly we were told that chloroform lowers the blood-pressure ; that this in itself shows that its administration is dangerous ; and that occasionally the fall is so unexpected and capricious as to produce sudden death by stoppage of the heart. The Hyderabad Commission has shown (1) that the lowering of the blood-pressure, which chloroform and all anæsthetics cause when efficiently administered, is in itself a harmless event, if the respiration alone be attended to and taken as a guide, and if the administration be stopped when the patient is fully anæsthetised ; and (2) that the sudden falls of pressure which the Glasgow Committee asserted are dangerous, and attributed to chloroform, are due to stimulation of the vagus, and, by slowing the circulation, are a safeguard against overdosing. The Hyderabad Commission has further proved that all irregularities in the fall of the blood-pressure and in the circulation under chloroform, including such an irregularity as dilatation of the heart, which never occurs when chloroform is administered properly, are due to improper administration, with irregular breathing and insufficient air.

There is no loophole of escape for the opponents of principles which Syme long ago showed to be essential for safety in the administration of chloroform. Tracings 3 and 4 of Experiment No. 186 of March 6th, 1890, were not produced accidentally, but are examples of what

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[* This statement is open to correction.]

† Black in the diagram.

happens in every case of chloroform inhalation without exception, whether in man or in animals, where the breathing is perfectly regular. Any one who is taught to give chloroform in the right way can obtain the same sequence of events clinically, which the tracings pourtray experimentally ; and I undertake to produce them invariably in any laboratory or operating room in the world. My students do it here every day, and if we can do it anybody can do it. The irregularities in the tracings of the Glasgow Committee, and Professor Macwilliam's more recent bogey of dilatation of the heart, are due to obstruction of the circulation in the lungs through interference with, or irregularity of, the respiration. The proof of this is furnished by the experimenters themselves. The Glasgow Committee state that in their experiments the anæsthetic was administered in a cloth, *saturated with chloroform*, held over the face, *i.e.*, with insufficient air. In Professor Macwilliam's experiments the chloroform was pumped into the lungs with bellows. Professor Macwilliam states that only a certain percentage of chloroform, 1 to 5 per cent., was allowed to get into the air, in the experiments he performed in this manner. Before we can accept his conclusions, however, he is bound to demonstrate that air containing from 1 to 5 per cent. of chloroform, blown into the trachea with bellows, is incapable of producing obstruction of the circulation or respiration in the lungs. Obstructed circulation in the lungs and a rapidly falling blood-pressure are more than enough to account for the dilatation of the whole heart, which occurred in his experiments and which he wrongly attributed to the direct action of chloroform. It is amusing to contrast Professor Macwilliam's statements regarding the danger of dilatation from the direct action of chloroform on the heart in the laboratory with Dr. James Dunlop's clinical experience, as narrated in *The Lancet* of September 27th, 1890, that in typical cases of death from the direct action of chloroform the heart is empty and flaccid. Perhaps Mr. Alexander Wilson will assert that Professor Macwilliam's statements "are only a little fuller and more complete" than Dr. Dunlop's, and that they are "not incompatible," which is the view he takes of Erichsen's teaching that the pulse should always be taken as a guide, and Syme's teaching that it should never be taken as a guide in chloroform administration.

I am satisfied to leave the question of chloroform administration to the judgment of the readers of the Medical Chronicle. I have always understood that, next to saving life, the prevention and relief of pain are among the most important duties of our profession. According to the teaching of the Hyderabad Commission, pain may safely be prevented and relieved with chloroform by any intelligent and properly taught medical practitioner; and there can be no question that every medical man ought to be able to give a dose of chloroform with as much precision and certainty as a dose of morphine, or of any other poison. The practical outcome of the disastrous teaching of the Glasgow Committee, backed up by Professors Wood and Macwilliam, is that the relief of pain by chloroform is to be handed over to specialists, who alone are to administer it, though their own declarations and statistics show that they cannot give it with safety. Exactly in proportion as this teaching gains ground the bulk of the profession suffers loss in credit and in pocket, and the advantages chloroform confers are most seriously restricted and curtailed. There is no more necessity for the anæsthetist, with his inhaler and other apparatus, than for a hypodermic morphia injectionist, or other poisonist. If the anæsthetist is to be tolerated hereafter at all, it must be because he is a man of pleasant presence and cheerful countenance, who knows how to comfort and re-assure the patient until chloroform oblivion is secured.

## ARTICLE REPRINTED FROM *THE MEDICAL CHRONICLE* OF FEBRUARY 1891.

By JOHN A. MACWILLIAM, M.D.,

Professor of the Institutes of Medicine in the University of Aberdeen.

In the *Medical Chronicle* of January, 1891, Surgeon-Major Lawrie, in an article on the Hyderabad Chloroform Commission, makes some comments on certain results obtained by me in a recent investigation on the action of chloroform and ether, published in the *British Medical Journal*, October 11th, 18th, and 25th, 1890. In my paper I described the frequent occurrence of dilatation of the whole heart under the influence of anæsthetic doses of chloroform. Such dilatation was not due to changes in the pulmonary circuit or to a fall of arterial pressure.

Dr. Lawrie urges that the cardiac dilatation *was* due to obstruction of the circulation in the lungs, or to this along with a fall in the blood-pressure. He says: "The irregularities in the tracings of the Glasgow Committee and Professor Macwilliam's more recent bogey of dilatation of the heart, are due to obstruction of the circulation in the lungs through interference with or irregularity of the respiration." And later: "Obstructed circulation in the lungs and a rapidly-falling blood-pressure are more than enough to account for the dilatation of the whole heart, which occurred in his experiments, and which he wrongly attributed to the direct action of chloroform."

Now this hypothesis of Surgeon-Major Lawrie's can be briefly and conclusively disposed of. There is an abundance of decisive evidence available. First, as regards the alleged influence of obstruction of the circulation in the lungs in causing dilatation of the heart. In my paper in the *British Medical Journal* I stated the fact that such an explanation was incompetent to explain the cardiac condition, since the *whole organ* was dilated in my experiments. Dilatation of the right side of the heart might conceivably (if there were no evidence to the contrary) be accounted for by the presence of pulmonary obstruction. But dilatation of the left side (*e.g.*, the left auricle) could clearly not be accounted for in the same way, since pulmonary obstruction necessarily diminishes in large measure the flow of blood into the left auricle and this part becomes small and collapsed. This seemed so obvious to me that I did not discuss the matter at any great length.

I shall now state briefly some facts in regard to Surgeon-Major Lawrie's hypothesis of obstructed circulation in the lungs:—

(1) There is no proof whatever of the occurrence of pulmonary obstruction from the administration in mammals of anæsthetic doses of chloroform sufficiently diluted with air, as

was the case in my experiments, the amount of chloroform vapour in the air never exceeding four per cent.

(2) On the other hand, there is decisive proof that pulmonary obstruction was *not* present in my experiments. Obstruction of the circulation would necessarily cause distension of the pulmonary artery as well as, and indeed earlier than, distension of the right heart. The pulmonary artery would become large and tense in consequence of the increased peripheral resistance to the outflow of blood from that vessel. Then the increased tension would react upon the right heart and cause distension there also. But in my experiments *there was no distension of the pulmonary artery during the administration of chloroform.* The vessel did not become distended ; it was soft and compressible, and the tension within it was low. It is quite certain then that pulmonary obstruction could not have been the cause of the dilatation even of the right side of the heart.

(3) Even if pulmonary obstruction had been present—as was not the case—such obstruction could not have produced the condition which I described, or anything at all similar to it.

I have on various occasions purposely brought about a condition of pulmonary obstruction by mechanical means with a view to studying the features of this condition. This I did by injecting into one of the great systemic veins some particulate substance, *e.g.*, lycopodium powder, which would cause embolism of the pulmonary vessels, and so obstruct the circulation in the lungs. When such a substance is injected into the vein it rapidly passes through the right heart into the pulmonary vessels, speedily causing more or less extensive and sudden obstruction of the pulmonary circulation, according to the amount and suddenness of the injection.

The effects resulting from the plugging of the minute pulmonary vessels induced in this way are very noteworthy, and illustrate clearly the consequences of obstruction of the circulation in the lungs. The pulmonary artery swells up and becomes largely distended, while at the same time the vessel feels hard and tense to the touch ; the pressure within the vessel is greatly elevated. The right ventricle and the right auricle then partake in the condition of distension, being unable to discharge their contents in the normal fashion in face of the greatly augmented resistance in front. The lungs become pale and anæmic. Meanwhile, it is important to observe that the left auricle becomes small and collapsed-looking, in consequence of the obstacle offered to the passage of the blood through the lungs. Strong respiratory efforts, with convulsions of the asphyxial type (accompanied by spasmodic contraction of the systemic arteries) supervene in consequence of the stoppage of the respiratory purification of the blood ; the medullary centres become violently stimulated by the venous character of the blood. Death speedily follows.

It is unnecessary to enter closely into a comparison, or rather contrast, of the typical illustration of pulmonary obstruction here afforded, and the condition of cardiac dilatation which I have described as occurring under the influence of chloroform. It is obvious that obstruction of the circulation in the lungs produces changes strikingly different from those that result from

**the action of chloroform; and that the hypothesis of pulmonary obstruction may at once be dismissed as entirely insufficient and inapplicable, as far as an explanation of the cardiac dilatation occurring under chloroform is concerned.**

Seeing that it is easy to dispose of the question of pulmonary obstruction in regard to the effects of chloroform upon the heart, I shall now advert briefly to the possibility of cardiac dilatation being dependent on a rapidly falling blood-pressure, as alleged by Dr. Lawrie. This is a matter which I carefully considered in my paper, and in regard to which I was able to state definitely that the cardiac dilatation is *not* due to a fall of blood-pressure. This is conclusively proved by the following facts :—" (1) Dilatation of the heart does not by any means always run parallel to the fall of pressure which ordinarily results from the administration of chloroform. Sometimes there is a very marked fall of pressure, with little or no dilatation of the heart ; while at other times the heart begins to dilate before the pressure begins to fall. (2) Moreover, the heart in some instances begins to dilate during the temporary *rise* of pressure which at times precedes the fall. (3) Further, there may be distinct dilatation of the heart without any change of pressure at all. (4) Lastly, it can easily be shown that a simple fall of pressure, equal in amount to that which ordinarily occurs under chloroform, induced by means which do not directly affect the heart (*e.g.*, section of a vaso motor nerve, hæmorrhage, *etc.*), does not cause dilatation of the organ as chloroform does."

It is clear, then, that the dilatation of the heart brought about by chloroform cannot be due to obstruction of the circulation in the lungs, or to a fall of systemic blood-pressure ; it must result from a depressing influence exerted by the anæsthetic on the organ (1) directly, or (2) through the vagus nerves.

Finally, as this depressing influence, leading to dilatation, is readily manifested after section of both vagi, it is obvious that chloroform must act upon the heart directly.

I cannot help feeling that even a comparatively slight amount of *actual observation* of the features and results of a simple lowering of the blood-pressure and of *real* pulmonary obstruction would have prevented Surgeon-Major Lawrie from putting forward and adhering to such a hypothesis as I have here discussed,—a hypothesis at once insufficient, absolutely untenable, and in conflict with large and somewhat elementary facts in the physiology of the circulation.

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## ARTICLE BY SURGEON-MAJOR EDWARD LAWRIE.

[*Reprinted from the " Medical Chronicle " of May* 1891.]

CONTROVERSIES on medical topics, even where great principles are involved, are apt to degenerate into mere word-quibbling or wrangling. I shall endeavour in the present article on chloroform to conduct the discussion between the Hyderabad Commission and Professor Macwilliam into a channel of agreement which will interest the whole profession, by whose judgment the question in dispute must ultimately be decided.

I have stated (vide *The Lancet* for June 21st, 1890), and still state that a probable cause of cardiac dilatation, when chloroform is pumped into the lungs with bellows, is pulmonary stasis. This statement is made on the authority of the greatest physiologists in the world—Professors Michael Foster and Gaskell. In 1879 the Glasgow Committee attributed cardiac dilatation to pulmonary obstruction; in 1890 they ascribed it to paralysis of the heart; and now Professor Macwilliam asserts that it must result from a depressing influence exerted directly by the anæsthetic on the heart, presumably as a muscle. It is remarkable that while the Glasgow Committee and Professor Wood are as much opposed to Professor Macwilliam as we are, they are all three antagonistic to the Hyderabad Commission, but they are only linked by unenlightened fear of chloroform.

The profound and irreconcilable differences of opinion beween the Glasgow Committee in 1879 and in 1890, and between the Glasgow Committee and Professor Macwilliam, are of no importance in this connection, as there are serious sources of error in Professor Macwilliam's experiments and conclusions, which reduce all his arguments about dilatation of the heart to insignificance.

Professor Macwilliam gives four facts, which he says, prove conclusively that cardiac dilatation is not due to fall of blood-pressure. They are as follows :—

"(1) Dilatation of the heart does not by any means always run parallel to the fall of pressure which ordinarily results from the administration of chloroform. Sometimes there is a very marked fall of pressure, with little or no dilatation of the heart; while at others the heart begins to dilate before the pressure begins to fall. (2) Moreover, the heart in some instances begins to dilate during the temporary *rise* of pressure which at times precedes the fall. (3) Further, there may be a distinct dilatation of *the heart without any change* of pressure at all. (4) Lastly, it can easily be shown that a *simple fall* of pressure, equal in amount to that which ordinarily occurs under chloroform, induced by means which do not directly affect the heart (*e.g.*, section of a vaso motor nerve, hæmorrhage, &c.) does not cause dilatation of the organ as chloroform does."

These four facts, if facts they be, distinctly prove that Professor Macwilliam's results with chloroform are not uniform. Sometimes one effect is produced, sometimes another. I shall demonstrate later that the irregularity of his results was caused by the irregularity of, or interference with, the respiration and the normal oxygenation of the blood in his experiments.

From Professor Macwilliam's first fact it is clear that dilatation is not failure of the heart function, because it bears no relation to the blood-pressure. Fact two shows that dilatation is probably the result of the rising pressure with which it occurs, and this in its turn is the result of the contraction of the small arteries stopping the outflow of blood. Professor Macwilliam's third fact, that " there may be a distinct dilatation of the heart without any change of pressure at all " is paradoxical. It is impossible to believe that blood-pressure can be maintained with a failing heart, seeing that the heart is the only source of pressure. If heart dilatation can really occur without fall of pressure, it must be a natural condition and harmless. We have no right to expect the heart to do anything more than maintain pressure in the aorta. That is its sole function, and if pressure be maintained the heart cannot be said to fail. Professor Macwilliam's description of rhythmical dilatation is also very suspicious. If the dilatation were due to the chloroform action on the heart muscle it would assuredly go on increasing to the end. Professor Macwilliam assumes dilatation to exist because his base line rises whereas a rise of base line might be produced by anything that raised the heart as a whole. The entire arrangement is so artificial that it is very difficult to accept any of his results, or to believe that he can make all his levers and tambours, six in number, work in the perfect way he describes, and at the same time that he can watch the condition of the pulmonary artery so narrowly (in the cat !) as to be able to assert that it was soft and compressible, and that the pressure within it was low. The fourth fact is meaningless. No one would expect the heart to dilate with loss of blood. In bleeding, the blood is removed from the arteries and stored outside the body. By chloroform poisoning blood is not lost, but is removed from the arteries, the pressure falling in consequence, and stored in the veins, perhaps in the heart itself. Granting, however, that Professor Macwilliam's cardiac dilatation is real, its depressing influence is a pure assumption and is opposed to the very facts on which he relies to prove his case.

The truth is that the Glasgow Committee in 1879, and Professor Macwilliam in 1890, drew conclusions from an inadequate number of experiments. The Glasgow Committee made their observations on less than half a dozen animals ; Professor Macwilliam made his on 75 cats. Just as the Hyderabad Commission and Professor Macwilliam have shown that the fall of pressure, which the Glasgow Committee considered dangerous in chloroform administration, is harmless, and may be a safeguard, so some future observer will show that Professor Macwilliam's cardiac dilatation is not only harmless but is a safeguard also. It would not be surprising if Professor Macwilliam's dilatation of the heart turns out in this way to be an important physiological discovery of the same kind as Dr. Bomford's discovery of the safeguard action of the vagus nerve. It is likely enough that the whole vascular system is relaxed by chloroform, including the walls of the heart. Under such circumstances the safeguard action of dilatation may consist in an arrangement to allow the blood naturally to collect in the heart, which is the best place for it, ready to be pumped into the aorta at a moment's notice, and so restore the pressure. Suppose the heart is completely relaxed by chloroform, as the systemic muscles are, it will go on again if respiration is kept up. In any case there is no danger clinically from cardiac dilatation in chloroform inhalation. Clinical results cannot be accepted only when they favour the view that chloroform is dangerous. Probably as much chloroform is used in India as in the whole of Europe, and we have no evidence of dilatation of the heart, as a dangerous effect of chloroform, from one end of the year to the other.

The Hyderabad Commission did not discover the key to the safe administration of chloroform, until they had made observations upon 565 animals and had reached their 148th manometer experiment. The tracings of this experiment are produced here, and ought to be compared with the tracings of the experiment of March 6th, 1890, which were so beautifully engraved in the January number of the *Medical Chronicle*. In experiment No. 148 chloroform was at first given freely on a cap, held close over the animal's mouth and nose. Every time Dr. Brunton gave the signal to give chloroform, the cap was immediately held over the face, and the animal struggled, and held its breath, and gasped. The blood-pressure fell with extreme rapidity and irregularity, on each of the four occasions, when chloroform was given in this way between 2h. 50m. 0s. and 3h. 11m. 0s. After the fourth administration, at 3h. 11m. 0s., I warned the chloroformist to hold the cap some distance from the face, and allow the animal to breathe regularly. This was done at the next inhalation at 3h. 17m. 0s., and the contrast with the previous administrations was so marked, that Dr. Brunton thought the chloroformist was playing tricks, and said "You are not giving the chloroform." There was no struggling, holding of the breath, or irregular breathing, and no irregularity either of the fall of blood-pressure, or of the circulation. On the contrary, the tracing was perfectly regular, and the observation at Fick 7 shows absolute regularity of the pulse. These observations were repeated during the experiment with the same result.

From experiment 148, when the clue was obtained, until the end of the research, the Hyderabad Commission gradually worked out to a satisfactory conclusion the problem of the safe administration of chloroform. We discovered that it is possible to give chloroform in such a way as to produce invariably uniform results. It was found that, in chloroform inhalation, if the breathing be kept perfectly regular, the fall of the blood-pressure, the action of the heart, and the circulation will be perfectly regular also ; and under these circumstances it is impossible to overdose the animal, unless the administration is continued after full anæsthesia is produced. Any irregularity in any of these particulars in chloroform inhalation indicates a corresponding irregularity of, or interference with, the respiration and the normal oxygenation of the blood; and it is difficult to imagine a greater irregularity of respiration than is involved in pumping chloroform into the lungs with bellows as was done in Professor Macwilliam's experiments. We were able to demonstrate that it is in the very highest degree dangerous to administer chloroform to an animal while the breathing is irregular, particularly if there be struggling or holding of the breath. When either of these untoward events occurs, the animal may be forced to gasp in chloroform at an abnormal rate, and irremediable overdosing may take place, without warning, at any moment.

The results arrived at by the Hyderabad Commission correspond precisely with those obtained clinically. I have now given chloroform for more than a year, on the lines indicated by the Commission, with absolute uniformity. In the human subject, as in animals, so long as the breathing is perfectly regular and the inhalation is stopped in good time, there is no danger whatever in pushing chloroform until full anæsthesia is produced. But it is excessively dangerous to administer chloroform to a struggling patient, or if the patient's breathing is irregular, or to give it in such a way as to make him hold his breath. Hence the rule—which is now enforced here without exception—that, if the patient struggles or breathes irregularly, or holds his breath, the chloroform cap must be entirely removed, and the patient must be made to take a breath of fresh air before the administration is proceeded with.

The Hyderabad Commission do not pretend to have done more with regard to chloroform than test its suitability and safety as an anæsthetic. They have shown how it can be administered with uniform safety, and that its administration is surrounded with natural safeguards ; and their experimental data correspond with clinical experience. The immunity from fatalities enjoyed by Syme and by hundreds of his pupils, in whose practice deaths from chloroform have been altogether exceptional, and amount to nothing like the London statistics of 1 death in every 1,236 administrations,* cannot be set aside as accidental or due to chance. Professor Macwilliam's present position is untenable; and his theory that dilatation of the heart is a danger of chloroform, and that therefore the pulse must be watched during its inhalation, has received its death-blow from one of his own supporters. In *The Lancet* for December 13th, 1890, Dr. Dudley Buxton describes a method of chloroform administration, in which feeling the pulse and taking it as a guide is an essential principle. This method causes, according to its author, general feebleness of the circulation with " distinct and unmistakable cardiac enfeeblement," which " is progressive, and likely, it has appeared to me in more than one case, to end in cardiac syncope unless prompt and vigorous measures were used." Dr. Buxton goes on to state that the anatomical counterpart of this faulty method is to be found in the relaxation and dilatation of the heart in Professor Macwilliam's experiments. It is probably true that Professor Macwilliam's experimental data correspond, as Dr. Dudley Buxton says they do, with " the procession of events which one has encountered again and again in the operating theatre," but there is little doubt that this agreement between the two is equally damaging to both, no less so to Professor Macwilliam's experimental methods than to Dr. Dudley Buxton's plan of giving chloroform.

The Hyderabad Commission takes its stand on the fact brought out by its own experiments, and since verified clinically, that the only way to keep the pulse regular under chloroform is to keep the breathing regular. This requires all the unceasing skill, care, and attention of the chloroformist. Chloroform is administered in practice by inhalation. If the breathing is regular the anæsthetic will be inhaled in regular quantities ; regular breathing constitutes in reality the only reliable means of measuring the dose ; and no capricious or irregular effects can be produced on the circulation or the pulse. If the breathing is irregular, the chloroform will be inhaled in irregular quantities, which will give rise to irregularities of the circulation and of the pulse, and danger will always be imminent. It is obvious that without special risk to the patient the pulse cannot be taken as a guide by which danger can be averted under chloroform. Failure of the pulse indicates heart-failure,† and is a sign, like pallor of the face and dilatation of the pupil, that a dangerous stage has already been reached; and danger in chloroform inhalation implies improper administration and overdosing, either from irregularity of the breathing or from the anæsthetic having been pushed too far. Those who take the pulse as a guide unconsciously allow " distinct and unmistakable cardiac enfeeblement " to occur, and then proceed to rescue the patient by " prompt and vigorous measures." The Hyderabad Commission seeks to entirely avert danger in chloroform administration by keeping the breathing regular, which is the only possible way of protecting the heart and circulation. In short, the object of the chloroformist must be to give chloroform in such a way that danger can never arise; and this object can be attained by any intelligent medical man who chooses to follow implicitly the teaching of the Hyderabad Commission. The matter can, however, very easily be brought to the proof. On the facts established by the Hyderabad Commission, I challenge the Glasgow Committee, or Professor Wood or Professor Macwilliam, to produce either an irregular trace or a dangerous dilatation of the heart with chloroform, before stoppage of the respiration, in any animal whose breathing is normal and regular throughout the administration.

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* *Vide* Mr. Roger William's letter in *The Lancet* of February 8th, 1890.

[† There is one exception to this rule. Failure of the pulse and sudden pallor may indicate inhibition of the heart's action through stimulation of the vagus. These symptoms are in themselves harmless, and are unquestionably a safeguard; but the mere fact that the safeguard action of the vagus is brought into play is dangerous, as it shows that the anæsthetic has been improperly administered with insufficient air ; and it demands the immediate employment of artificial respiration.]

## ARTICLE BY PATRICK HEHIR, M.D., F.R.C.S.E.

Surgeon, Bengal Medical Service; President, First Hyderabad Chloroform Commission.

*[Reprinted from the " Medical Chronicle " of June* 1891.]

THE various, discrepant and irreconcilable opinions recently expressed in medical periodicals regarding the question as to the right method of inducing chloroform anæsthesia in the human being, indicate that even the highest authorities on the subject entertain almost opposite views. These opinions have poured in from all quarters during the last twelve months, that is, since the publication of the Report of the Second Hyderabad Chloroform Commission. When we consider that it is this very difference of opinion in regard to the proper method of producing insensibility by inhalation of chloroform vapour (in other words, that it is the variations in the way of administering it) that lies at the foundation of the great disparity of mortality statistics from chloroform anæsthesia, we must admit that the question itself is a most momentous one, and one that might with credit to the profession be removed from its present equivocal basis.

The history of the origin of the Hyderabad Chloroform Commission is too well known to require any further explanation here beyond that of stating that the object in view was to show that the principles upon which the late Professor Syme administered chloroform were sound and reliable. We cannot be too frequently reminded as to what these principles are: " Free admixture of air with the vapour of chloroform, which is ensured by using some soft, porous material, as a towel or lint, presenting a large surface; no stint in the quantity of chloroform given; watch the respiration."* He considered that there was never any occasion to feel the pulse or examine the heart either before or during anæsthesia.

The main practical points brought out by the Hyderabad Chloroform Commissions, were: (1) that primary cardiac syncope during chloroformisation does not occur, and (2) that the respiration is the only safe guide to the effect of chloroform. In the report of the First Hyderabad Chloroform Commission we made the following statement: † " In no case did cardiac syncope occur. The Commission considers that it is impossible for chloroform vapour to kill a dog by acting primarily on the heart, and this holds good no matter in what doses or in what manner the anæsthesia is induced. Death from primary cardiac syncope never took place." Further, "With regard to the anæsthesia produced by chloroform in dogs, once insensibility is brought about, so long as respiration is kept up, there is no fear of a cumulative effect, the drug is rapidly eliminated. Interference with this eliminative process brings about changes in the respiratory mechanism, which ought to arouse suspicion and prepare us for artificial respiration. The keenest watchfulness of the respiration was indispensable from the beginning to the close of chloroformisation, if safety was to be guaranteed." I am thoroughly convinced that the same remarks hold good with regard to chloroform anæsthesia in the human being.

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* Vide *Lancet*, 1855, Vol. I., p. 55. † Vide *Lancet*, January 22, 1890.

These conclusions were arrived at from simple observation, and record of the phenomena occurring during the chloroformisation of dogs. They were adequately substantiated, supplemented and placed on a scientific foundation by the experiments of the Second Hyderabad Chloroform Commission, and have not, as far as I have been able to glean from the various discussions that have arisen on the question, been as yet successfully controverted.

Before the conclusions of the Second Hyderabad Chloroform Commission were made known, the fact that respiration is always affected before the heart caused the most scrupulous attention to be paid by the adherents of Syme's principles to the condition of the former during chloroform anæsthesia, and it was found that if the respiration ceased, which it occasionally did, there was always time to restore the patient by artificial respiration. Under this old system of administration, in all probability overdosing with the vapour not unfrequently took place, but the rapid elimination effected by the prompt artificial respiration, which was sometimes demanded, rendered the patient exempt from real danger. Surgeon-Major Lawrie has gone a step beyond his former teaching: he now removes the anæsthetic as soon as there is any impediment to the respiratory mechanism, beginning it again as soon as uniform breathing is re-established. He now insists on (1) chloroform being given in such a way that the respiration is regular throughout the administration, and (2) the anæsthetic being entirely removed, and the patient taking a breath of fresh air if any struggling, holding of the breath, or irregular respiration occurs. The observance of this simple practical rule is an absolute safeguard against overdosing with chloroform vapour, and with this plan of administration it is difficult to imagine a death from chloroform inhalation taking place otherwise than under a combination of fortuitous circumstances, or as a purely accidental event. Formerly the respiration was watched for warnings whereby danger might be arrested, now it is attended to in such a way that danger can never arise.

One great lesson to be learnt from the results of the Second Hyderabad Chloroform Commission's experiments is that regularity of the respiration is to be maintained throughout the administration of chloroform, and that a continuance of the anæsthetic during any interference with regular respiration is associated with danger. Previous to the announcement of the conclusions of the Second Commission, all those who give chloroform on the late Professor Syme's principles watched the respiration, knowing that when it became shallow, embarrassed, or ceased, danger was to be apprehended if prompt artificial respiration was not resorted to; but now, in addition to watching the respiration, it is incumbent on us to the utmost degree possible to keep it regular. The maintenance of this regularity of breathing is rightly insisted upon by Surgeon-Major Lawrie, and constitutes the key to the safe administration of the anæsthetic.

The two occasions on which I have experienced any danger in connection with chloroform administration were:—

(1) In the case of a child 2½ years of age on whom the operation of circumcision was being performed. In the middle of the operation he began vomiting, and during a violent inspiratory effort suddenly sucked a piece of guava about the size of an almond into the larynx. Respiration absolutely ceased, and were it not that I had the good fortune to hook the piece of guava out with the finger (a process which must have taken about half a minute to carry out), the case must have been fatal if an opening were not made into the larynx. The operation was performed about 9 a.m., the mother having been previously told that the child was to have nothing but a little milk that morning before the operation, and as a matter of fact she did not know that the child had stolen into the garden and partaken of guava. All through the period during which the lump of guava effectually blocked up the *rima glottidis*, my assistant informed me that the pulse beats were regular.*

(2) The second case was one of phlegmonous erysipelas of the scalp, into which latter incisions were to be made. The man (an old clerk in the Revenue Department of His Highness the Nizam's Government) was placed on the table, and my assistant was directed to give chloroform in the usual way. I was in an adjoining room about eight yards off, and told the assistant to send in word when the patient was anæsthetised. In about four minutes' time he rushed into the room and said that the respiration had stopped. I found that it had ceased. The patient was restored by artificial respiration. In this case, likewise, the pulse was to be felt during the whole period of the cessation of respiration. This accords with Professor Syme's principles, and with the experience of both Hyderabad Chloroform Commissions.

My personal experience with regard to the administration of chloroform is limited to about 1,[illegible]00 cases, yet in these, it has been given under almost every conceivable circumstance, but always according to the principles laid down by Professor Syme. In six cases I have kept patients continuously under chloroform for over twelve hours, in three of puerperal eclampsia, two of tetanus, and one of hydrophobia : and in one case—that of strychnine poisoning—for five hours. All these cases except that of hydrophobia, recovered. In my student days I was taught the administration of chloroform on Syme's principles, and I have consistently adhered to them during the last eleven years.

I have known Surgeon-Major Lawrie to give chloroform on these principles for the last fourteen years with perfect safety, and his experience in this respect is probably unique. The late Professor Syme's and Surgeon-Major Lawrie's uninterrupted series of cases without a single death form a monument of evidence in favour of the principles upon which they have administered this anæsthetic. The students under Surgeon-Major Lawrie's instructions give chloroform four or five times daily in the Afsul Gunj and Residency Hospitals, and I hazard the statement that they do so with a *savoir faire* that would be in no way discreditable to special anæsthetists.

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* A case precisely similar to this one, but occurring in an adult, and terminating fatally, was recorded in the *Madras Mail* in 1888. The coroner's verdict was death from " accidental causes ".

To my reasoning, the whole theory of safe chloroformisation might be summed up in the aphorism—*Use pure chloroform, watch the respiration and keep it regular.* This may appear to be a most dogmatic assertion, and coming from one with my meagre experience may tinge of presumption. Still, I have given chloroform for over eleven years; I have seen it given by Surgeon-Major Lawrie for many years, and I have worked with both the Hyderabad Chloroform Commissions, and may on these grounds claim to be in a position to express an opinion on the subject.

In conclusion, I would state that the more widespread the knowledge of the late Professor Syme's principles of chloroformisation, and the more universally they are put into practice, the greater will become the confidence of the profession in this much maligned anæsthetic, and the fewer, I believe, will be the deaths under chloroform from "accidental causes." To Surgeon-Major Lawrie the profession is indebted for his unswerving endeavours to restore chloroform to its proper position amongst anæsthetic agents.

## ARTICLE BY SURGEON-MAJOR EDWARD LAWRIE,

President of the Hyderabad Chloroform Commission.

[*Reprinted from the "British Medical Journal" of June 13th* 1891.]

THE *British Medical Journal* has performed a signal service to the Hyderabad Commission in placing before the profession Dr. Julliard's views on chloroform and ether. Dr. Julliard had a death with Chloroform. Then instead of learning to give it safely he took, apparently with great reluctance, to ether. He has now learned to give ether safely and minimises its disadvantages by giving morphia. People who have been taught to give chloroform with safety are not likely to follow his example. Moreover the leading article in the *Journal* of April 25th, 1891, is incomplete, and ought to have included the London statistics so opportunely brought forward by Mr. Roger Williams in *The Lancet* of February the 8th, 1890.

According to Dr. Julliard's statistics, deaths from chloroform amount to 1 in 3,258 and from ether to 1 in 14,987 administrations. According to Mr. Roger Williams the statistics of the London hospitals show that deaths from chloroform amount to 1 in 1,236 and from ether to, in 2,754 administrations. On the other hand, the statistics of chloroform administered on Syme's principles form an unbroken accord of inhalations from 1848 to 1891 without a death. The *British Medical Journal* regards Dr. Julliard's figures as a "most valuable statistical summary," but this summary would obviously be much more useful if it were accompanied by a description of the method of administration pursued in all the cases from which it is compiled. There are two distinct methods of chloroform administration in vogue. In one the pulse, as well as the respiration, is taken as a guide: in the other, the pulse is never, under any circumstances, taken as a guide, and it is manifestly unreasonable to compare the risks of ether and chloroform without stating with regard to chloroform which of these methods is employed. The importance of this point lies in the fact that there is not one case of death from chloroform recorded in which it is proved that the pulse was not taken as a guide; whereas in Syme's practice and in my own, where the pulse has never been taken as a guide, no death from chloroform has ever occurred. If the pulse is affected under chloroform it indicates chloroform poisoning either direct or through abnormal respiration. All the chloroformist has to produce is normal anæsthesia and of this the pulse can never be any test whatever. It is therefore positively dangerous and useless to take it as a guide. The following table places the available figures in a most striking light :—

*Mortality Statistics of Ether and Chloroform.*

| Anæsthetic employed. | Source of Statistics. | Period. | Number of deaths to administrations. |
|---|---|---|---|
| Chloroform ... ... | Julliard ... ... ... | Not stated ... ... | 1 to 3,258 |
| Ether ... ... ... | Julliard ... ... ... | Do. ... ... | 1 to 14,987 |
| Choroform ... | Bartholomew's Hospital (Roger Williams) ... ... | 10 years, 1878 to 1887... | 1 to 1,236 |
| Ether ... ... | Bartholomew's Hospital (Roger Williams) ... ... | Do. ... | 1 to 2,754 |
| Chloroform ... ... | Syme and Lawrie ... ... | 43 years ... ... ... | No death. |

If statistics are of any value, this table ought to carry conviction with it because it shows clearly that chloroform administered on Syme's principles is even less dangerous than ether administered in accordance with the most approved methods. But the Hyderabad Commission has no desire to institute further comparisons between them. All we say is, let anybody use ether who chooses, but if chloroform is to be employed, let it be given in the right way. Surgery cannot yet do without chloroform, and the only way to give it with invariable safety is to be guided, as Syme was, not by the circulation, but entirely by the respiration. What Dr. Julliard says about ether I can say *mutatis mutandis* about chloroform. During fourteen out of the seventeen months * that have elapsed since the Hyderabad Commission demonstrated that the key to the safe administration of chloroform consists in regular breathing, I have given chloroform several times daily. Not only have I not had any deaths, but I have met with no accident of any kind. I have not once had to do artificial respiration or to pull forward the tongue. Neither have I had to interrupt an operation in order to ward off any accident due to chloroformisation. There is no element whatever either of luck or of chance about these results. Any surgeon can administer chloroform without risk who will take the trouble to assure himself that the patient's breathing is normal and regular throughout the administration, and to stop the inhalation in good time, *i.e.*, directly full anæsthesia is produced. Statistics such as those of Dr. Julliard and Mr. Roger Williams, which are intended to show the danger of chloroform, are, as my table proves, susceptible of a very different interpretation. If they help to prove anything, it is that no anæsthetic is absolutely safe except chloroform administered on Syme's principles, and the more proof we have of this kind the better.

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* For three months I was absent on leave.—ED. L.

# LECTURE ON CHLOROFORM

## By Surgeon-Major EDWARD LAWRIE.

**Dr. Lawrie**, President of the Hyderabad Chloroform Commission, delivered a lecture on Chloroform in the Grant Medical College, Bombay, on Monday, June 29th, 1891 before a large assembly of the medical profession. Surgeon-General Pinkerton, who presided, briefly introduced the lecturer, saying he had much pleasure in introducing Dr. Lawrie, of the Bengal Service, to them. He had come there that day to tell them about his experiments at Hyderabad. It was unnecessary for him (the chairman) to say much, as they would have read all that had been said on the subject, and Dr. Lawrie, he believed, was very anxious to give those who had taken it up a fair field and no favour. Thoroughly imbued with the results of his observations, he naturally wished to have the sympathy of all medical men, especially of all medical men in India. After Dr. Lawrie had given his views, he would be very glad if they discussed any point upon which they might differ, or which might not be clearly understood.

Dr. Lawrie, who was received with applause, then proceeded with his lecture. He said:

"Mr. President, Ladies and Gentlemen,—Few people who have read the preliminary report of the Hyderabad Commission are aware of the real liberality and public spirit displayed by the Nizam's Government in issuing the Commission. His Highness's Government paid Dr. Lauder Brunton a fee of £1,000. The Government also paid the whole of the expenses of the Commission, including a complete set of invaluable photographs of the tracings of the experiments, and entertained the Commission royally during their stay in Hyderabad. In addition to this, Government defrayed the whole of the expenses connected with the report. Many people I have conversed with appear to think that it gave me an immense amount of trouble, and that I had to employ a great deal of persuasion to get the Commission appointed. Gentlemen, I never spoke to the Nizam but once about the appointment of the Commission. The Nizam is one of the cleverest men in India, and directly I explained to him what was wanted he understood it exactly, and after a short conversation he said he would issue orders for the Commission. It is needless for me to say anything further regarding the extraordinary and unprecedented liberality and interest with which the Minister, Sir Asman Jah, and every member of His Highness's Government, carried those orders into effect.

"In 1855 Mr. Syme published a clinical lecture, in which he said: 'the points that we consider of the greatest importance in the administration of chloroform are, first, a free admixture of air with the vapour of the chloroform, to ensure which a soft porous material presenting a pretty large surface is employed, instead of a small piece of lint or any apparatus held to the nose. If this is attended to, the more rapidly the chloroform is given the better till the effect is produced. Then,—and this is a most important point—we are guided as to the effect, not by the circulation, but entirely by the respiration. You never see anybody here with his finger on the pulse while chloroform is given. So soon as the breathing becomes stertorous, we cease the administration.' The Hyderabad Commission does not pretend to have done more with regard to chloroform than prove that Syme's principles are right, and I hope to be able to lay the proof clearly before you this evening. Notwithstanding

Syme's teaching, two distinct methods of chloroform administration are, and always have been, practised. In one the pulse, as well as the respiration, is taken as a guide, and deaths have been numerous. In the other, the pulse is never, under any circumstances, taken as a guide, and in Syme's practice and in my own, extending over a period of forty-three years, no deaths from chloroform have ever occurred. In 1879 the British Medical Association appointed a Committee in Glasgow to experiment with anæsthetics. They made a limited number of observations on dogs and rabbits with self-recording apparatus, and they showed by tracings that chloroform lowers the blood-pressure, and that sometimes during chloroform inhalation the fall is unexpected and capricious. The lowering of the blood-pressure the Glasgow Committee called the normal, but dangerous, effect of chloroform. The sudden falls with slowing of the heart indicated, in their opinion, a source of extreme danger. The tracing and report of the experiments of the Glasgow Committee appeared to demonstrate (1) that lowering of the blood-pressure is due to weakening of the heart's action ; (2) that this effect is peculiar to chloroform and is never observed with ether ; and (3) that death may occur at any time during chloroform inhalation by sudden stoppage of the heart. The terms 'primary and secondary chloroform syncope' crept into use and settled themselves in the nomenclature of medicine. The effect of the Glasgow Committee's report was enormous, far-reaching, and disastrous. No blame is to be attached to the Glasgow Committee, because they published their tracings, and anybody is at liberty to form his own opinion about them. Having accepted them, we made ourselves just as much responsible for them as they were. No one stopped to inquire whether a general fall of blood-pressure is harmless or dangerous, or whether the fall of blood-pressure registered in their experiments was due to direct weakening of the heart or not. Common sense was cast to the winds, and two erroneous principles became established on a pseudoscientific basis. One is, that the pulse *must* be felt during chloroform administration, and the other is, that ether is not only a safe anæsthetic, but is, under all circumstances, a cardiac stimulant. To such an amazing pass have these two principles been pushed that the public have been made to believe that feeling the pulse is a safeguard in chloroform administration, and that it requires a specialist to do it. On the other hand, the blind and unfounded belief in ether as a cardiac stimulant has led to its employment hypodermically in cases of chloroform-poisoning, and even in ether-poisoning itself. The clinical experience and statistics which the *Lancet* called for after the Hyderabad Commission's report was published, have only brought out the curious fact that all chloroform deaths now-a-days are possibly the result of hypodermic injections of ether, which is always resorted to as soon as possible after symptoms of danger appear. It would be just as wrong to give subcutaneous injections of chloroform in ether-poisoning as to give subcutaneous injections of ether in chloroform-poisoning.

"The report of the Glasgow Committee was accepted by the whole profession as practically final. I remember, when it was published in 1879, I felt at first as if Syme's position was no longer tenable. As time went on, however, and it was found that chloroform could still be given on Syme's principles with the same safety as before, I began to realise that there must be some flaw in the experiments or conclusions of the Glasgow Committee. Accordingly in 1888 Sir Asman Jah, the Minister of Hyderabad, sanctioned the appointment of a Commission with Surgeon P. Hehir as president, to make further experiments with chloroform. Dr. Hehir's Commission proved that in death from chloroform the breathing always stops before the heart, and in his report he made the original observation that as long as breathing is kept free there is no danger of a cumulative effect, and chloroform can be given with absolute safety. This conclusion was not accepted by the profession. It led to a tardy discussion, and in 1889 His Highness the Nizam appointed another Commission. Through the enlightened liberality of His Highness's Government, the *Lancet* sent out Dr Lauder Brunton to represent it on the Commission, and at the same time the Government of India deputed Surgeon-Major Bomford. His Highness's Government was represented by Surgeon P. Hehir, Dr. Rustamji, and myself. No better selection could possibly have been made by the *Lancet* than Dr. Lauder Brunton. He came out to India thoroughly convinced that chloroform paralyses the heart. During the whole time the Commission worked, he tried to stop the heart in every conceivable way, and failed. The Commission found that shock and chloroform are not associates but incompatibles. It is impossible to produce cardiac syncope, or even vaso-motor depression, by any form of shock in any stage of chloroformisation. When it was found impossible to produce any effect upon the heart by any form of shock under chloroform, Dr. Bomford suggested that we should give chloroform and then stop the heart by electrical irritation of the vagus nerve, and see what happened. This was done, and what happened was this. We found that so far from being a danger under chloroform, stopping the heart actually proved to be a safeguard against poisoning, by slowing the circulation and delaying the conveyance of chloroformed blood to the brain and spinal cord.

"The next phase of the Commission's research consisted in demonstrating that the so-called normal fall of the blood-pressure under chloroform, which the Glasgow Committee considered dangerous, and the unexpected and capricious falls which they thought excessively dangerous are in themselves harmless. The cause of the gradual falls I shall refer to at a later period. We were able to prove that the sudden and capricious falls are due to inhibition or stoppage of the heart from stimulation of the vagus nerve from abnormal respiration or over-dosing, and are as much a safeguard against poisoning with chloroform as I have just shown that the inhibition caused by electrical stimulation of that nerve is." Dr. Lawrie here handed round and distributed among the audience several photographs of the tracings of the Glasgow Committee's experiments, and of No. 148 of the Hyderabad Commission's experiments, and pointed out that the tracings of the Glasgow Committee's observations A & C are identical with observations 8, 9, 11, 15, and 16 in Fick tracings ii & iii, which were produced by chloroform and by ether. Dr. Lawrie explained that the Glasgow trace could always be reproduced by (*a*) stimulation of the vagus and by (*b*) asphyxia ; and he further added that it was frequently produced accidentally during the experiments of the Hyderabad Commission, by the animal holding its breath or struggling (*vide* experiment No. 148 and many others), or by stoppage of the respiration from over-dosing with chloroform.

"In the third phase of the Commission's labours, it was distinctly proved that uniform results can always be obtained with chloroform, provided it is sufficiently diluted to allow of regular normal breathing throughout the whole administration. As long as these conditions are fulfilled, the fall of the blood-pressure is regular, and the heart's action and the pulse are invariably regular also. We proved that any irregularity of the heart or of the pulse under chloroform is a certain sign of chloroform-poisoning, either from direct overdosing or from overdosing through abnormal or irregular respiration. Of normal anæsthesia with regular breathing and without poisoning, which is, in practice, all the chloroformist has to produce, the pulse can never be any test whatever. It is only a test of irregular breathing or of overdosing, and it is, therefore, no less dangerous than useless to take it as a guide.

" Such is a very brief outline of the conclusions and results arrived at by the Hyderabad Commission. It would be an insult to your common sense to exaggerate them, or to pretend that without further amplification they define the whole position with regard to chloroform. Undoubtedly they go a very long way towards doing so, but the old question still remains unanswered. That question is, does chloroform affect the heart directly? This question is inextricably mixed up with the cause of the fall of the blood-pressure under chloroform, and in order to answer it the cause of this fall must now be determined. If chloroform in any way whatever directly weakens the heart, it is clear that its administration can never be free from risk. Clinically I know that it does not weaken the heart, because I or anybody else can produce normal anæsthesia with chloroform, with regular breathing and without overdosing, entirely without risk. This fact, however, will never convert the profession; and though I have often been annoyed during the past eighteen months because our results have been so little acted upon, I now see that it is unreasonable to expect the profession to accept them until the fall of blood-pressure under chloroform is fully and satisfactorily accounted for. Last year, I had the honor of an interview with Professors Michael Foster and Gaskell at Cambridge, and they examined the tracings of the Hyderabad Commission's experiments. Professor Foster looked over them for a long time without saying a word. At last he stated his opinion that the one thing brought out by the whole of the tracings is that the blood-pressure is lowered by chloroform, as it is by all anæsthetics, and that this appears to indicate danger to the heart; but he added, 'against this we must remember that when a poison is introduced into the system, everything that happens afterwards is of necessity in the nature of a safeguard.' Apart from vagus stimulation, there are two possible ways of lowering the blood-pressure: one by weakening the heart, and the other by vaso-motor dilatation—dilatation of the small arteries. If the lowering of the blood-pressure under chloroform were due to direct weakening of the heart, it is clear that a patient under chloroform would be in a gradually increasing state of syncope and that the fall of blood-pressure would be in itself a danger, and this is opposed to the facts that there is no danger in normal anæsthesia and that there is absolutely no resemblance between anæsthesia and syncope. But if the fall is due to relaxation of the vaso-motor system of muscles and consequent dilatation of the small arteries, it must be absolutely harmless; and in accordance with Foster's principle it ought to be a safeguard. If we carefully consider the effects of chloroform, we find they are as follow: As consciousness is abolished under chloroform, first the systemic muscles are relaxed and sent to sleep, and then the respiratory muscles, and during this time the blood-pressure is falling—regularly and gradually if the respiration is normal and not interfered with. The relaxation of the systemic and respiratory muscles is due to narcosis, or sending to sleep, of the central nervous system, and not to any direct action of chloroform upon the muscles themselves. After the muscles of respiration are relaxed and respiration ceases, the heart can no longer obtain nourishment. The animal then gradually dies, and the heart stops beating. The next point with regard to this most interesting subject is that the work of the heart is not kept going either by a cardiac centre in the brain or spinal cord, or by the ganglia in its own substance. At first this may sound a little like heresy, but the tendency of modern physiology, and of modern physiologists headed by Gaskell, is to show that the heart is a self-acting muscle, and a recent discovery by Dr. Arthur Meigs of Pennsylvania gives ground for the belief that this conception is literally true. The fibres of ordinary muscle derive their nutrition by absorption from delicate capillary blood-vessels which run between them. Dr. Meigs has demonstrated that the muscular fibres of the heart have a circulation peculiarly their own. He has shown that in the heart a capillary blood-vessel enters into the substance of the muscle fibre, and there can be no doubt that each fibre is nourished, and at the same time stimulated to action, by the blood flowing in its interior. The muscular fibres of which the heart is composed are, if this view is correct, automatic hearts in miniature, and the heart itself is thus a self-acting organ stimulated to rhythmic beating throughout the whole of life by the very blood which nourishes it. Dr. Meigs's discovery explains and accounts for the vigour of the circulation in the heart as well as the high state of its nutrition. It also shows that each successive beat of the heart is produced by the beat immediately preceding it. We have seen that the heart is independent of the central nervous system, and there is no more ground for supposing that chloroform acts directly upon its muscular tissue than that it acts directly upon any other muscular tissue. It is, therefore, a fair inference that the heart can never be directly affected by chloroform. We now come to the last link in the chain. Chloroform narcosis affects first the brain, and then the spinal cord. The vaso-motor centre is situated in the upper part of the spinal cord, in the *medulla oblongata*, and it must be narcotised like the other nerve centres. The vaso-motor muscles are thus relaxed, and the blood-pressure falls. As the relaxation of the vaso-motor system is not vital, the fall of blood-pressure is harmless."

Dr. Lawrie now handed round photographs of the tracings of the Hyderabad Commission's experiment No. 178, Ludwig II and Fick I, and of Experiment No. 186 of March 6th, 1890, Ludwig I, II, III, and IV. Referring to the Ludwig tracing II of experiment No. 178, he said:

"In this observation chloroform administration was commenced at 2h. 51m. 40s. The blood-pressure fell in the usual way, and at 2h. 54m. 35s. the breathing and heart both stopped. A reading was taken on the Fick manometer, and the complete arrest of the heart's action for over a minute is well shown at Fick 13. This reading is remarkable as showing that the blood-pressure had already fallen as low as it could get before the heart stopped, and it did not fall further after this happened. The fall must, therefore, have been due to vaso-motor dilatation and not to weakening of the heart. I must now ask you to examine the tracings of Experiment No. 186 of March 6th, 1890. Ludwig I shows the harmless effect of sudden lowering of the blood-pressure at 2h. 17m. 50s. and at 2h. 48m. 25s. ; the animal was quite out of chloroform at these times, and it was not even being administered. Chloroform administration was commenced with plenty of air and regular breathing at 2h. 18m. 35s. The gradual and regular fall of blood-pressure is very well shown in this observation. The breathing stopped at 2h. 24m. 5s., and electrical irritation of the vagus was begun at 2h. 24m. 15s., and continued until 2h. 25m. 10s. The only apparent effect of the vagus stimulation was to slow the heart without lowering the blood-pressure. Artificial respiration was not employed, and the animal recovered rapidly.

"Ludwig II.—In this observation chloroform administration was commenced simultaneously with electrical irritation of the vagus at 2h. 35m. 50s. The blood-pressure fell suddenly almost to zero. This was followed by a pause and then two slow beats of the heart. The pulse trace then slowly returned, and the pressure gradually rose to a little more than half its former height, and then gradually fell again as it does in normal chloroform administration. The pulse remained slow. The respiration ceased entirely at 2h. 37m. 15s. The vagus irritation, which was begun at 2h. 35m. 50s., was continued until 2h. 39m. 5s., when it was stopped, and normal breathing at once recommenced without any further steps being taken to revive the animal. In this observation the effect of the vagus irritation was to suddenly lower the blood-pressure and to slow the pulse. The stoppage of the respiration is to be particularly noted and kept in mind with reference to the next observation in Ludwig III.

"In Ludwig III, chloroform inhalation with normal breathing was commenced at 2h. 53m. 35s. The blood-pressure fell gradually and regularly. The breathing stopped at 2h. 55m. 45s., but it was not recorded till 2h. 56m. 0s. in order to make sure that it had quite stopped. Electrical irritation of the vagus was commenced at 2h. 56m. 10s, and continued till 2h. 57m. 25s. The blood-pressure fell suddenly and remained low, with slow action of the heart for one minute and fifteen seconds. The animal recovered spontaneously without artificial respiration or any other treatment. (If Ludwig IV is now examined, it will be seen that the chloroform observation of Ludwig III was repeated. When the breathing stopped, the animal was left entirely alone and he died.) The question that arises about the animal's recovery in the experiment recorded in Ludwig III is, what was the recovery due to? It must be noted that the vagus was not divided. The visible effect of the irritation of the entire vagus was to lower the blood-pressure which was already dangerously (?) low, and slow the pulse. It might be objected that the irritation of the uncut vagus stimulated the respiratory centre, and so saved the animal's life. But if this might be said about Ludwig III, it might equally fairly be said about Ludwig II that the irritation of the vagus at 2h. 35m. 50s. saved the dog's life by stopping the respiration, which would be absurd, as the irritation of the vagus cannot be a safeguard, or save life, by stopping the respiration in one case and exciting it in another. If lowering the blood-pressure were dangerous and due to weakening of the heart, it is clear that the stimulation of the vagus at 2h. 56m. 10s., when the animal's breathing had stopped and the pressure was already perilously (?) low, was the very worst and most dangerous expedient we could have adopted, as it at once caused a further fall of the blood-pressure, and kept it down nearly to zero for considerably over a minute. So far from being a dangerous procedure, however, it saved the dog's life ; and this fact alone is sufficient to upset the hitherto accepted belief that general lowering of the blood-pressure is dangerous, or that it is due in chloroformisation to weakening of the heart. The last point to notice with reference to Ludwig tracing III is, that it is evident that the pulse beats increased in force from the time chloroform was administered at 2h. 53m. 35s. till the commencement of the artificial fall of the blood-pressure at 2h. 56m. 10s., and that at the end of this fall at 2h. 57m. 25s. the pulse tracing was at first pretty large, and that as the animal's pressure rose during recovery it gradually got smaller and smaller. If the lowering of the blood-pressure were due to weakening of the heart, the reverse of this ought to have occurred, the pulse ought to have become smaller and smaller up to the stop, and afterwards begun by being very small and gradually increased in size as the animal recovered.

"Finally, with reference to the fall of the blood-pressure in all the tracings exhibited this evening, it is to be observed that the sudden (unexpected and capricious) falls, which the Glasgow Committee considered exceptionally dangerous, did not actually prove to be so in their experiments (their animals did not die) ; and have been demonstrated in ours to be a safeguard. If the *sudden* falls are a safeguard, *a fortiori* the *gradual* falls, which occur in normal chloroform administration, are a safeguard also.

"We have now the proof, why normal chloroform anæsthesia is free from danger. It is because chloroform never, under any circumstances, directly affects the heart, and that the fall of blood-pressure is due to vaso-motor dilatation, and, so far from being a risk, is in all cases, if not harmless, a safeguard. I have only one word to add in conclusion. If you examine the tracings you will see what splendid work was done by Dr. Bomford for the Commission. Each tracing is a work of art ; but each one was completed by Dr. Bomford, and everything that occurred was written on it as the drum revolved at the time of the experiment. Nothing was added to any one of them after the experiment was over, so that nothing was trusted to any one's memory. In the second place, Dr. Bomford discovered the safeguard action of the vagus nerve, and this proved to be the key to the safe administration of chloroform. In everything I have written since the Commission ended the physiology is a reproduction of Dr. Bomford's views, generally in his own words ; and I feel proud to think that work so permanent and lasting as that of the Hyderabad Chloroform Commission was carried to a successful conclusion mainly through the instrumentality of a member of that Service to which I and so many of my hearers have the honour and the happiness to belong—the Indian Medical Service." (Loud applause.)

Dr. Wellington Gray then said he had been for a long time connected with the J. J. Hospital, and had seen many more than 10,000 administrations of chloroform. Since the sitting of the Chloroform Commission he had watched the administrations of chloroform with increased care and interest. No doubt there was much to be discovered, and much to be inquired into yet, but, from his own clinical experience, he believed the views and conclusions of the Hyderabad Commission were not far from the truth.

Dr. Arnott said he was glad to find the views of Dr. Lawrie were the same as he had been taught by Professor Lister twenty-five years ago; in that Sir Joseph Lister insisted that the great safeguard in the giving of chloroform was the dilution of the chloroform, and that the giving of the chloroform in a concentrated form, such as a closed cone, closely applied over the nose and mouth of the patient, was not a safe method of administration; but that some method of dilution must be adopted to the amount which Dr. Lawrie had quoted, and which, he thought, was about the amount quoted by Professor Lister some twenty-five years ago. He had no doubt that the administration of diluted chloroform was as safe as Dr. Lawrie had said.

Dr. Hatch next explained his experiences in chloroform administration. During ten or twelve years he had only known of two deaths, and one of those he had some doubts about before administering. In these cases the chloroform was administered with the closed cone, which it was not necessary to place close to the nose.

Dr. Blaney said he had been Coroner of Bombay for fifteen years, and every death that had been reported as having occurred from chloroform or from any other anæsthetic, had been inquired into by him, as it was his duty to do, but he said it was to the credit of the medical profession of Bombay that they stood behind no other city he knew of in the successful administration of chloroform. Those reports he had received of deaths from chloroform, he did not consider worth the paper they had been written on. Chloroform, during the time he had been Coroner, must have been administered in Bombay many thousands of times, but there were only two deaths during that time which really resulted from chloroform or anæsthetics.

Some other gentlemen reported their experiences, saying they had had no deaths from chloroform, and expressing the opinion that there was no danger from chloroform if properly administered and diluted. At the same time it was necessary, some thought, to watch the pulse as well as the respiration.

Dr. Lawrie said that before the Commission he used to watch the respiration for signs whereby danger might be arrested, but that since the session of the Commission he did not merely watch the respiration, but insisted on its being kept absolutely regular throughout the whole administration so as to produce normal inhalation and normal anæsthesia.

Surgeon-General Pinkerton proposed a hearty vote of thanks to Dr. Lawrie for coming there and delivering that lecture; and to H. H. the Nizam for affording the means of research.

The motion was heartily responded to, and the meeting separated.

# PART IX.—CLINICAL RESULTS.

[By the PRESIDENT.]

The system of note-taking in chloroform administration, now in force in the Afzulgunj Hospital, has been developed very gradually since the completion of the experiments of the Commission. In the first 272 cases in which an accurate record of chloroformisation was kept, the notes were taken by students. The average time to produce anæsthesia in these cases, which include children and adults, was 3m. 44s. In 82·9 per cent. abolition of the corneal reflex, in 16·8 per cent. stertorous breathing, and in one case complete relaxation of the muscles was the first sign of full anæsthesia. There was no instance of respiratory embarrassment or of accident or delay of any kind due to over-dosing, and the patient was never allowed to inhale the anæsthetic while the breathing was in any way irregular.

From Case No. 273 the notes were taken by the surgeon. The system of administration and note-taking was improved by the adoption of suggestions made by two of the most distinguished surgeons in Europe during the discussion on anæsthetics in Glasgow. Sir George Macleod stated that in all his public operations he administers chloroform himself until full anæsthesia is produced, Dr. William MacEwen said that during chloroform inhalation every one of the patient's respirations should be registered on the chloroformist's tympanum. Effect was given to both these principles. Sir George Macleod's method has been practically followed by the surgeon himself taking notes of every case of chloroform administration in the presence of the clinical class, while the administration has been entrusted to students as before. In this way the responsibility for the whole case rests with the surgeon as it always ought to rest, unless we are to go back to the days of barber surgery when the surgeon simply did as he was told ; at the same time the students, without exception, have the opportunity of becoming skilful and safe chloroformists. Throughout the inhalation the chloroformist carries out Dr. MacEwen's rule to the letter and every one present is kept informed of the rate of the patient's respirations by an assistant who stands near the chloroformist and taps the table after every completed breath the patient takes.

The administration of chloroform in the Afzulgunj Hospital is now conducted in the following manner. When all is ready for the operation the surgeon gives the signal to begin the inhalation. The method of administration is described on page 286. While it is proceeding no one is allowed to do anything in the operating room, not even speak, and everybody's attention is thus concentrated on the administration until the patient is fully anæsthetised. From the moment the inhalation is commenced until anæsthesia is produced, the surgeon records every event, even of the most trivial kind, with the exact time of its occurrence. This is in strict conformity with Dr. Bomford's plan of recording on the tracings everything, as it took place, during the manometer experiments of the Commission. Each entry is notified to the class as the surgeon records it, and after the operation is finished, and the patient has been removed, one of the students is made to describe from memory what happened during the inhalation. His description is compared with the record, and finally, the record is read out to the class.

In the cases detailed it will be remarked that the respiratory pauses, which are sometimes referred to as "apnœa" and sometimes as "holding breath," were systematically followed by one breath of pure air. This requires a word of explanation. During chloroform administration the patient usually holds his breath from apnœa, or owing to over-concentration of the vapour, or from increasing narcosis of the respiratory centre. Holding the breath from apnœa, which occurs in the early stages of the inhalation, is generally due to rapid breathing, and when the patient begins to breathe again he does so quietly and without gasping. Holding the breath from over-concentration of the vapour of chloroform or from sluggishness of the respiratory centre, though a natural safeguard, leads to asphyxia, and when the patient is compelled to breathe again he gasps. It is not always possible to determine the cause of holding the breath; but it is evident that as the patient can take in no chloroform as long as he is not breathing, it does not waste time to remove the chloroform cap from his face until he breathes again. When the patient does breathe again, however, it makes all the difference in the world whether the chloroform cap is over his face or not. If it is, and he gasps, an over-dose may be taken in or still further irregularity of the respiration may be produced; in either case the result is abnormal inhalation. But if the first inspiration after holding the breath, or after any abnormality of the breathing, consists of a breath of pure air, all danger is avoided. Normal respiration is restored; and normal respiration alone is compatible with normal inhalation and normal anæsthesia.

## *Cases of chloroform administration at the Afzulgunj Hospital.*

### August 22nd, 1891.—No. 273.

Temperature of operating room 73·7° F. Healthy Mahomedan male ætat 29. Disease—hydrocele. Operation—excision of sac. Chloroformed by student Krishtiah at 8h. 13m. 30s. Anæsthesia complete at 8h. 17m. 45s.; operation commenced at 8h. 18m. 0s.; finished and patient removed from table at 8h. 44m. 30s. Retched and brought up a little frothy mucus at 8h. 41m. 55s.

*Observations.*

| | H. | M. | S. | |
|---|---|---|---|---|
| A. | 8 | 13 | 30. | —Chloroform on cap; patient blowing hard. |
| B. | 8 | 14 | 20. | —Apnœa; cap entirely removed till 8h. 14m. 45s.; one breath fresh air. |
| C. | 8 | 14 | 45. | —Chloroform; regular breathing. |
| D. | 8 | 16 | 10. | —Struggling; regular breathing. Struggling continued till 8h. 16m. 50s. when one breath of fresh air was given, and the struggling ceased. |
| E. | 8 | 17 | 0. | —Snoring; at 8h. 17m. 30s. the snoring was accompanied by noisy expiration. |
| F. | 8 | 17 | 45. | —Breathing stertorous; stop chloroform. |
| G. | 8 | 17 | 55. | —Cornea insensitive. |
| H. | 8 | 18 | 0. | —Operation commenced. |
| I. | 8 | 44 | 30. | —Operation completed. |

In this and in all subsequent cases, unless it is expressly stated to the contrary, the anæsthesia was continued until the completion of the operation and the application of the dressings, in the usual way, by giving chloroform from time to time.

### August 22nd, 1891.—No. 274.

Temperature of room 76·3°F. Shurfun Bee (female infant), ætat 1 year. Disease—abscess. Operation—incision. Chloroformed by student Miss Williams at 9h. 3m. 0s. Anæsthesia complete at 9h. 4m. 10s. Chloroform stopped and operation finished at 9h. 7m. 15s.

*Observations.*

H. M. S.

A. 9 3 0.—Chloroform on cap ; crying and struggling ; breathing regular.

B. 9 4 10.—Cornea insensitive ; kept under chloroform till the dressing was applied, and the child was removed from the table at 9h. 7m. 15s.

August 22nd, 1891.—No. 275.

Temperature of room 76·3° F. Male Mahomedan child, Mahomed Sahib, ætat. 1 year. Disease—wound. Explored under chloroform and sutured. Chloroformed by student Miss Williams at 9h. 12m. 55s. Under at 9h. 13m. 40s. Administration stopped and operation finished at 9h. 18m. 5s.

*Observations.*

H. M. S.

A. 9 12 55.—Chloroform on cap ; crying and struggling ; breathing regular.

B. 9 13 10.—Holding breath ; chloroform cap removed.

C. 9 13 20.—Gasped fresh air ; chloroform again.

D. 9 13 30.—Regular breathing.

E. 9 13 40.—Cornea insensitive ; kept under chloroform till 9h. 18m. 5s.

August 24th, 1891.—No. 276.

Temperature of room 78·1° F. Well-nourished Hindoo male, Krishna Row, ætat. 44. Disease—leprosy. Operation—nerve splitting and cleaning. Chloroformed by student Krishtiah at 7h. 40m. 0s. Full anæsthesia at 7h. 45m. 10s. Chloroform stopped at 8h. 8m. 5s.

*Observations.*

H. M. S.

A. 7 40 0.—Chloroform on cap ; blowing gently.

B. 7 42 30.—While more chloroform was being poured into the cap the patient took three breaths of pure air.

C. 7 43 30.—Noisy expiration.

D. 7 44 15.—Breathing irregular ; one breath of pure air.

E. 7 45 0.—Snoring.

F. 7 45 10.—Stertorous breathing ; chloroform stopped. Kept under till the operation was finished at 8h. 8m. 5s.

### August 24th, 1891.—No. 277.

Temperature of room 73·1° F. Weakly Mahomedan male, Shaik Jamal, ætat. 30. Disease—abscess. Operation—incision. Chloroformed by student Krishtiah at 8h. 13m. 50s. Full anæsthesia at 8h. 18m. 50s. Administration stopped at 8h. 24m. 30s.

*Observations.*

| | H. | M. | S. | |
|---|---|---|---|---|
| A. | 8 | 13 | 15. | —Chloroform on cap ; blowing very rapidly into the cap, 68 times a minute. |
| B. | 8 | 15 | 0. | —Cap close to face. |
| C. | 8 | 15 | 10. | —Breathing irregular ; one breath of fresh air. |
| D. | 8 | 16 | 0. | —Regular breathing, 24 respirations a minute. |
| E. | 8 | 17 | 10. | —Snoring. |
| F. | 8 | 17 | 30. | —Two breaths of fresh air while more chloroform was added. |
| G. | 8 | 18 | 45. | —Noisy expiration. |
| H. | 8 | 18 | 50. | —Cornea insensitive ; chloroform stopped. Administration afterwards continued till 8h. 24m. 30s. |

### August 24th, 1891.—No. 278.

Temperature of room 73·1° F. Healthy Hindoo male, Ramiah, ætat. 20. Disease—needle in the buttock. Operation—excision. Chloroformed by student Krishtiah at 8h. 31m. 50s. Full anæsthesia at 8h. 36m. 5s. Administration stopped at 8h. 40m. 0s.

*Observations.*

| | H. | M. | S. | |
|---|---|---|---|---|
| A. | 8 | 31 | 15. | —Chloroform on cap ; blowing rapidly 52 times a minute. |
| B. | 8 | 31 | 50. | —Talking. |
| C. | 8 | 32 | 5. | —Cap close to the face. |
| D. | 8 | 32 | 10. | —Apnœa ; cap removed. |
| E. | 8 | 32 | 20. | —One breath of fresh air. Cap re-applied. Regular breathing 24 times a minute. |

| | H. | M. | S. | |
|---|---|---|---|---|
| F. | 8 | 33 | 10. | —More chloroform on cap; took two breaths of fresh air. |
| G. | 8 | 34 | 0. | —Moving legs and arms. |
| H. | 8 | 34 | 15. | —Holding breath and struggling; cap removed. |
| I. | 8 | 34 | 45. | —Gasped fresh air; cap re-applied; regular breathing. |
| J. | 8 | 34 | 50. | —Talking. |
| K. | 8 | 35 | 10. | —Noisy expiration. |
| L. | 8 | 36 | 5. | —Cornea insensitive; chloroform stopped. Administration afterwards continued till 8h. 40m. 0s. |

August 24th, 1891.—No. 279.

Temperature of room 73·1° F. Healthy Hindoo male, Kissensingh, ætat. 40. Disease—necrosis of the lower jaw. Operation—removal of dead bone. Chloroformed by student Krishtiah at 8h. 52m. 0s. Full anæsthesia at 8h. 59m. 55s.; the administration was slow on account of bleeding into the mouth. (A probe had been introduced into the sinus before the operation was decided on.)

*Observations.*

| | H. | M. | S. | |
|---|---|---|---|---|
| A. | 8 | 52 | 0. | —Chloroform on cap; blowing regularly. |
| B. | 8 | 53 | 5. | —Cap brought close to face. |
| C. | 8 | 54 | 30. | —More chloroform; two breaths of fresh air. |
| D. | 8 | 55 | 40. | —Noisy expiration. |
| E. | 8 | 56 | 20. | —Coughing violently; one breath of fresh air. |
| F. | 8 | 57 | 20. | —More chloroform on cap; two breaths of fresh air. |
| G. | 8 | 59 | 55. | —Cornea insensitive; administration continued till 9h. 4m. 55s. |

August 24th, 1891.—No. 280.

Temperature of room 86·5° F. Healthy Hindoo female, Lutchoo, ætat. 40. Disease—Ovarian cysts, Operation—double ovariotomy. Chloroformed at the Residency Hospital at 1h. 0m. 0s. by student Miss Williams. Full anæsthesia at 1h. 4m. 30s. Continued until the operation was finished at 1h. 59m. 0s.

*Observations.*

| | H. | M. | S. | |
|---|---|---|---|---|
| A. | 1 | 0 | 0. | —Chloroform on cap ; blowing regularly 36 times a minute. |
| B. | 1 | 0 | 40. | —Cap close to face. |
| C. | 1 | 0 | 55. | —Noisy expiration ; 32 respirations a minute. |
| D. | 1 | 1 | 25. | —Took three breaths of pure air while chloroform was added to the cap. |
| E. | 1 | 2 | 0. | —Holding breath ; cap entirely removed. |
| F. | 1 | 2 | 15. | —Gasped pure air ; chloroform again. |
| G. | 1 | 2 | 40. | —Holding breath, cap entircly removed. |
| H. | 1 | 2 | 55. | —Gasped pure air ; chloroform again. |
| I. | 1 | 3 | 40. | —Talking ; regular breathing. |
| J. | 1 | 3 | 50. | —Regular breathing 24 times a minute. |
| K. | 1 | 4 | 30. | —Cornea insensitive ; stop chloroform. Continued afterwards until the operation was finished at 1h. 59m. 0s. |

August 25th, 1891.—No. 281.

Temperature of room 74·2° F. Healthy European boy, F. C. Harding, ætat. 13. Disease—abscess of the neck. Operation—incision. Chloroformed by student Abdullah Khan at 7h. 55m. 10s. Anæsthesia complete at 7h. 59m. 55s. ; administration stopped at 8h. 10m. 30s.

*Observations.*

| | H. | M. | S. | |
|---|---|---|---|---|
| A. | 7 | 55 | 10. | —Chloroform on cap ; blowing gently and crying. |
| B. | 7 | 56 | 20. | —Cap brought close to the face. |
| C. | 7 | 56 | 45. | —Calling out. |
| D. | 7 | 57 | 45. | —Took two breaths of fresh air while more chloroform was added to the cap. |
| E. | 7 | 58 | 0. | —Regular breathing. |
| F. | 7 | 59 | 35. | —Snoring. |
| G. | 7 | 59 | 55. | —Cornea insensitive ; anæsthesia afterwards kept up till 8h. 10m. 30s. |

### August 25th, 1891.—No. 282.

Temperature of room 74·2° F. Healthy Mahomedan, Mahomed Bux, ætat. 56. Disease—stricture of the urethra and perineal abscess. Operation—incision, and dilatation of the stricture with Holt's dilator. Chloroformed at 8h. 12m. 40s. by student Krishtiah. Full anæsthesia 8h. 16m. 45s. Anæsthesia continued till the operation was finished at 8h. 37m. 5s.

*Observations.*

| | H. | M. | S. | |
|---|---|---|---|---|
| A. | 8 | 12 | 45. | —Chloroform on cap ; blowing rapidly. |
| B. | 8 | 13 | 0. | —Coughing. |
| C. | 8 | 14 | 10. | —Holding breath ; cap removed till **8h. 14m. 22s.** |
| D. | 8 | 14 | 20. | —Gasped fresh air ; chloroform again ; regular breathing. |
| E. | 8 | 14 | 40. | —Struggling till 8h. 15m. 15s. ; regular breathing. |
| F. | 8 | 15 | 15. | —Irregular breathing. |
| G. | 8 | 15 | 20. | —One breath of fresh air ; regular breathing. |
| H. | 8 | 16 | 30. | —Stertorous breathing ; chloroform stopped. |
| I. | 8 | 16 | 45. | —Cornea insensitive ; kept under afterwards until the operation was finished at 8h. 37m. 5s. |

### August 25th, 1891.—No. 283.

Temperature of room 78·4° F. Healthy Native Christian, Francis, ætat. 51. Disease—stricture of the urethra and extravasation of urine. Operation—perineal section and incisions. Chloroformed by student Krishtiah at 8h. 54m. 30s. Full anæsthesia at 8h. 59m. 15s. ; administration stopped at 9h. 3m. 40s.

*Observations.*

| | H. | M. | S. | |
|---|---|---|---|---|
| A. | 8 | 54 | 30. | —Chloroform on cap ; blowing gently. |
| B. | 8 | 55 | 15. | —Cap brought close to the face. |
| C. | 8 | 55 | 30. | —Moaning. |
| D. | 8 | 55 | 45. | —Slight struggling ; regular breathing. |
| E. | 8 | 56 | 10. | —Moving hands and arms. |
| F. | 8 | 56 | 30. | —Breathing irregular ; one breath of fresh air. |

H. M. S.

G. 8 56 50.—Breathing irregular again ; one breath of fresh air.

H. 8 57 30.—More chloroform poured into cap ; took two breaths of fresh air.

I. 8 58 30.—Talking.

J. 8 59 0.—Rapid regular breathing.

K. 8 59 50.—Cornea insensitive ; administration afterwards continued till 9h. 3m. 40s.

### August 25th, 1891.—No. 284.

Temperature of room 78·4° F. Weakly ill-nourished child, ætat. 1 year, Mahomed Saheb. Disease—abscess. Operation—incision. Chloroformed by student Ismail Khan at 9h. 54m. 0s. Anæsthesia complete at 9h. 55m. 45s. Administration stopped at 10h. 1m. 30s.

*Observations.*

H. M S.

A. 9 54 0.—Chloroform on cap ; regular breathing.

B. 9 54 30.—Two breaths of fresh air given without any particular reason.

C. 9 55 0.—Regular breathing, 32 a minute.

D. 9 55 45.—Cornea insensitive ; cap removed. Administration continued till 10h. 1m. 30s.

### August 26th, 1891.—No. 285.

Temperature of room 74·7° F. Healthy Hindoo female, Rungoo, ætat. 30. Disease—whitlow. Operation—incision and removal of necrosed phalanx. Chloroformed by student Miss Williams at 8h. 20m. 0s. Full anæsthesia at 8h. 24m. 15s. Administration ended at 8h. 26m. 55s. The breathing became stertorous fifteen seconds after the chloroform was stopped when the cornea became insensitive.

*Observations..*

H. M. S.

A. 8 20 0.—Resisting and breathing irregularly ; struggled violently and cried till 8h. 20m. 50s.

B. 8 21 10.—Gasping noisy inspirations.

C. 8 21 15.—One breath of pure air.

H. M. S.

D. 8 21 20.—Regular breathing.

E. 8 22 30.—Moaning.

F. 8 23 0.—Talking incoherently.

G. 8 24 15.—Cornea insensitive ; chloroform stopped.

H. 8 24 25.—Snoring.

I. 8 24 30.—Stertorous breathing ; the anæsthesia was maintained until the operation was finished at 8h. 26m. 55s.

This case is an example of residual chloroform in the lungs deepening the anæsthesia after removal of the chloroform from the face and discontinuance of the inhalation. What happens in these cases is shown in many of the experiments of the Commission, and is especially well marked in No. 64 on the Ludwig tracings, just after Fick readings 11 and 17. (*Vide* Experiment No. 64, page 53.)

### August 26th, 1891.—No. 286.

Temperature of room 74·7° F. Sickly Mahomedan male, Umar Khan, ætat. 33. Disease—sinus. Operation—incision. Chloroformed at the Afzulgunj Hospital by student Krishtiah at 8h. 30m. 45s. Anæsthesia complete at 8h. 36m. 30s. Continued till 8h. 41m. 55s.

### *Observations.*

H. M. S.

A. 8 30 45.—Chloroform on cap ; gently blowing, 24 times a minute.

B. 8 32 15.—Cap close to the face ; struggled. Regular breathing.

C. 8 32 50.—Breathing irregular ; one breath of fresh air ; regular breathing.

D. 8 33 15.—Moving left arm.

E. 8 33 50.—While more chloroform was being added, took two breaths of pure air.

F. 8 34 30.—Regular breathing, 24 times per minute.

G. 8 35 20.—Moaning ; noisy expiration. While more chloroform was being added took two breaths of pure air.

H. 8 36 30.—Cornea insensitive; snoring ; cap removed; administration continued afterwards until 8h. 41m. 5s.

In this case after the patient had been removed one of the most intelligent students was made to describe from memory what had happened during the administration. He could only recall very imperfectly and inaccurately what he had seen a few minutes before. For example, he was under the impression that full anæsthesia had been produced in four minutes instead of six. The experiment was repeated in every case subsequently, with the result that, though the by-standers were always warned to pay particular attention with a view to giving evidence afterwards, in no single instance could the witness give anything like an accurate description of what occurred during the administration of the anæsthetic. The fact that it is extraordinarily difficult to remember precisely what takes place when every thing is normal during an administration, shows how entirely unreliable and worthless statements made from memory by " the competent witnesses who have recorded human deaths under anæsthesia"[*] must be, when they are obscured by the shock of a patient dying, and by the excitement of frantic efforts to restore animation.

### August 26th, 1891.—No. 287.

Temperature of room 77·7° F. Sickly Hindoo male, Gowkari, ætat. 22. Disease—abscess. Operation—incision. Chloroformed at the Afzulgunj Hospital by student Krishtiah at 8h. 58m. 10s. Full anæsthesia at 9h. 5m. 55s. Continued until 9h. 8m. 35s.

*Observations.*

| | H. | M. | S. |
|---|---|---|---|
| A. | 8 | 58 | 10.—Chloroform on cap : blowing gently, 32 times a minute. |
| B. | 8 | 59 | 20.—Cap brought close to face. |
| C. | 9 | 0 | 10.—Holding breath ; cap entirely removed. |
| D. | 9 | 0 | 30.—Gasped pure air ; cap re-applied. |
| E. | 9 | 1 | 0.—Regular breathing, 24 a minute. |
| F. | 9 | 1 | 20.—While more chloroform was being poured on cap took two breaths of fresh air. |
| G. | 9 | 2 | 45.—Regular breathing, 24 a minute. |
| H. | 9 | 3 | 20.—While more chloroform was being added, took three breaths of fresh air. |
| I. | 9 | 3 | 25.—Noisy expiration. |
| J. | 9 | 3 | 45.—Raising arms. |

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[*] Vide Professor Wood's Address on Anæsthesia—page 268.

| | H. | M. | S. |
|---|---|---|---|
| K. | 9 | 4 | 30.—Irregular breathing ; one breath of fresh air. |
| L. | 9 | 5 | 0.—Struggling. Regular breathing. |
| M. | 9 | 5 | 15.—Talking incoherently. |
| N. | 9 | 5 | 55.—Cornea insensitive ; chloroform stopped ; continued afterwards till 9h. 35m. 0s. |

August 27th 1891.—No. 288.

Temperature of room 74·5° F. Delicate Mahomedan female, Hoossain Bee, ætat. 30. Disease—abscess. Operation—incision. Chloroformed at the Afzulgunj Hospital by student Krishtiah at 8h. 25m. 0s. Full anæsthesia at 8h. 34m. 30s. Continued until 8h. 38m. 50s.

*Observations.*

| | H. | M. | S. |
|---|---|---|---|
| A. | 8 | 25 | 0.—Chlorofrom on cap ; breathing regularly. 32 times a minute. |
| B. | 8 | 26 | 0.—Cap brought close to face. |
| C. | 8 | 27 | 0.—One breath of fresh air. |
| D. | 8 | 28 | 10.—While more chloroform was being added, took two breaths of pure air. |
| E. | 8 | 28 | 30.—Breathing regular, 32 a minute. |
| F. | 8 | 31 | 10.—Holding breath ; cap entirely removed. |
| G. | 8 | 31 | 20.—Gasped fresh air ; regular breathing. |
| H. | 8 | 31 | 50.—Snoring ; while more chloroform was being added took one breath of fresh air. |
| I. | 8 | 33 | 35.—Noisy expiration. |
| J. | 8 | 34 | 30.—Cornea insensitive ; stopped chloroform ; continued afterwards until the operation was finished at 8h. 38m. 50s. |

August 27th, 1891.—No. 289.

Temperature of room 74·5° F. Healthy Hindoo boy, Jutta Shunker, ætat. 14. Disease—cleft ear. Operation—plastic operation. Chloroformed at the Afzulgunj Hospital by student Krishtiah at 8h. 50m. 30s. Anæsthesia complete at 8h. 57m. 15s. ; continued until 9h. 1m. 30s.

*Observations.*

H. M. S.

A. 8 50 30.—Chloroform on cap ; regular breathing, 34 in a minute.

B. 1 51 30.—Cap brought close to face ; regular breathing, 24 in a minute.

C. 8 52 40.—While more chloroform was being added, took two breaths of pure air.

D. 8 53 5.—Moved left leg.

E. 8 54 0.—While more chloroform was being poured on cap, took three breaths of pure air.

F. 8 54 10.—Retching.

G. 8 54 45.—Vomiting food ; cap removed.

H. 8 55 25.—Chloroformed again.

I. 8 55 35.—Struggling ; one breath of pure air.

J. 8 56 0.—Regular respirations, 28 in a minute.

K. 8 57 15.—Cornea insensitive ; stopped chloroform ; continued afterwards until the operation was finished at 9h. 1m. 30s.

August 27th, 1891.—No. 290.

Temperature of room 78·3° F. Weakly Hindoo male, Nanuklan, ætat. 30. Disease—cystitis. Operation—catheterism. Chloroformed by student Krishtiah at 9h. 14m. 50s. Full anæsthesia at 9h. 18m. 15s. The breathing became stertorous 45 seconds after the chloroform cap had been removed, and the inhalation had been discontinued on account of the cornea being insensitive. Vomited a small quantity of mucus and bile at 9h. 24m. 0s. Before the administration was commenced the patient was in great terror of having a catheter passed and resisted violently before chloroform was given.

*Observations.*

H. M. S.

A. 9 14 50.—Chloroform on cap ; blowing noisily through the closed lips.

B. 9 16 0.—Noisy expiration.

H. M. S.

C. 9 16 15.—Cap close to face.

D. 9 17 0.—Holding breath. Cap removed.

E. 9 17 10.—One breath of fresh air. Chloroform again.

F. 9 17 30.—Noisy expiration.

G. 9 18 15.—Cornea insensitive ; chloroform stopped.

H. 9 19 0.—Breathing stertorous. No chloroform was administered after 9h. 18m. 15s.

This is another example of anæsthesia deepening after stoppage of the administration owing to the absorption of residual chloroform in the lungs. (*Vide* Experiment No. 64, page 53).

### August 27th, 1891.—No. 291.

Temperature of room 78·3° F. Weakly Mahomedan male, Syed Jalaluddin, ætat. 28. Disease—abscess. Operation—incision. Chloroformed by student Mahomed Abdul Ganni at 10h. 1m. 40s. Full anæsthesia at 10h. 7m. 50s. Administration stopped 10h. 12m. 15s.

*Observations.*

H. M. S.

A. 10 1 50.—Chloroform on cap ; regular breathing, 36 a minute.

B. 10 2 45.—Cap close to the face, slight struggle, not breathing regularly.

C. 10 4 0.—Regular breathing, 24 per minute.

D. 10 5 25.—Noisy expiration.

E. 10 5 30.—Took two breaths of fresh air, while more chloroform was poured on to the cap.

F. 10 5 45.—Noisy expiration.

G. 10 5 55.—Snoring.

H. 10 6 0.—Cap removed, chloroformist unnecessarily alarmed about the breathing which was quite regular.

I. 10 6 15.—Administration continued ; regular breathing.

J. 10 7 25.—Snoring.

K. 10 9 30.—Cornea insensitive ; administration afterwards continued till 10h. 12m. 15s.

### August 28th, 1891.—No. 292.

**Temperature of room 74·2° F. Unhealthy feverish Hindoo, Ramiah, ætat. 30. Disease—suppurating hydrocele. Operation—incision. Chloroformed at the Afzulgunj Hospital by student Krishtiah at 8h. 33m. 50s. Anæsthesia complete at 8h. 39m. 30s.; continued until 8h. 43m. 50s.**

*Observations.*

| | H. | M. | S. | |
|---|---|---|---|---|
| A. | 8 | 33 | 50. | —Chloroform on cap; rapid blowing, 44 a minute. |
| B. | 8 | 34 | 50. | —Cap brought close to the face. |
| C. | 8 | 35 | 0. | —Apnœa; cap entirely removed. |
| D. | 8 | 35 | 20. | —One breath of fresh air; regular breathing. Chloroform again. |
| E. | 8 | 36 | 40. | —While more chloroform was being added took three breaths of fresh air. |
| F. | 8 | 37 | 20. | —Regular rapid breathing, 40 a minute. |
| G. | 8 | 37 | 40. | —Struggling; regular breathing. |
| H. | 8 | 37 | 50. | —One breath of fresh air; stopped struggling. |
| I. | 8 | 38 | 30. | —While more chloroform was added took five breaths of fresh air. |
| J. | 8 | 39 | 0. | —Noisy expiration and struggling. |
| K. | 8 | 39 | 20. | —Stertorous breathing. Chloroform stopped. Continued afterwards till 8h. 43m. 50s. Vomited bile. Temperature 102° F. in the mouth before the operation. |

### August 28th, 1891.—No. 293.

Temperature of room 74·2° F. Very weak and emaciated Mahomedan male, Mahomed Bekan Khan, ætat. 76. Disease—senile gangreen of the foot. Operation—partial amputation. Chloroformed at the Afzulgunj Hospital by student Krishtiah at 9h. 0m. 0s. Anæsthesia complete at 9h. 4m. 30s.; continued until 9h. 8m. 30s.

*Observations.*

| | H. | M. | S. | |
|---|---|---|---|---|
| A. | 9 | 0 | 0. | —Chloroform on cap. |
| B. | 9 | 0 | 40. | —Rapid respirations, 40 a minute. |
| C. | 9 | 1 | 5. | —While more chloroform was being poured into the cap took 2 breaths of pure air. |

| | H. | M. | S. |
|---|---|---|---|
| D. | 9 | 1 | 35.—Struggling. One breath of fresh air. |
| E. | 9 | 2 | 35.—Talking. |
| F. | 9 | 3 | 5.—While more chloroform was being poured on to the cap, took two breaths of fresh air. |
| G. | 9 | 4 | 30.—Stertorous breathing. Chloroform was afterwards continued until the operation was completed at 9h. 8m. 30s. |

### August 28th, 1891.—No. 294.

Temperature of room 78·7° F. Weakly Hindoo male, Avayah, ætat. 30. Disease—abscess. Operation—incision. Chloroformed at the Afzulgunj Hospital by student Krishtiah at 9h. 18m. 0s. Anæsthesia complete at 9h. 25m. 20s., and continued until 9h. 29m. 15s.

*Observations.*

| | H. | M. | S. |
|---|---|---|---|
| A. | 9 | 18 | 0.—Chloroform on cap ; respirations 28 per minute. |
| B. | 9 | 19 | 10.—Cap brought close to the face. |
| C. | 9 | 19 | 25.—Regular breathing, 36 per minute. |
| D. | 9 | 19 | 35.—While more chloroform was being poured into the cap, two breaths of pure air were taken. |
| E. | 9 | 20 | 35.—Regular breathing, 17 per minute. |
| F. | 9 | 21 | 10.—Raising his legs. One breath of fresh air. |
| G. | 9 | 21 | 40.—While more chloroform was being poured into the cap, two breaths of pure air were taken. |
| H. | 9 | 22 | 20.—Struggling ; regular breathing. |
| I. | 9 | 23 | 25.—While more chloroform was being poured into the cap, three breaths of pure air were taken. |
| J. | 9 | 24 | 0.—Moaning. |
| K. | 9 | 25 | 0.—Snoring. |
| L. | 9 | 25 | 20.—Cornea insensitive. Stopped chloroform. The administration was afterwards continued until 9h. 29m. 15s. |

### August 28th, 1891.—No. 295.

Temperature of room 78·7° F. Young healthy Mahomedan male, Hussain Khan, ætat. 30. Disease—abscess. Operation—incision. Chloroformed at the Afzulgunj Hospital by Lady Superintendent Miss E. A. Lawrie at 9h. 44m. 0s. Anæsthesia complete at 9h. 47m. 55s. Continued until 9h. 50m. 30s.

*Observations.*

H. M. S.

A. 9 44 0.—Chloroform on cap ; blowing quickly, 30 times a minute.

B. 9 46 0.—While more chloroform was being poured into the cap, took three breaths of pure air.

C. 9 46 45.—Regular breathing, 24 a minute.

D. 9 47 5.—Snoring.

E. 9 47 30.—Deep snoring.

F. 9 47 50.—Stertor ; stopped chloroform.

G. 9 48 0.—Cornea insensitive. Administration was kept up until 9h. 50m. 30s.

### August 29th, 1891.—No. 296.

Temperature of room 74·2° F. Strumous Mahomedan child, Morad Sahib, ætat. 4. Disease—sinus. Operation—incision. Chloroformed at the Afzulgunj Hospital at 8h. 29m. 0s. by Miss E. A. Lawrie. Full anæsthesia at 8h. 31m. 30s.; continued until 8h. 37m. 5s.

*Observations.*

H. M. S.

A. 8 29 0.—Chloroform on cap ; crying but breathing regularly.

B. 8 29 55.—Cap close to face ; regular respirations, 24 a minute.

C. 8 30 45.—Coughing.

D. 8 31 10.—Noisy expiration.

E. 8 31 30.—Cornea insensitive and breathing stertorous simultaneously. Stop chloroform ; afterwards administration kept up till 8h. 37m. 5s.

## August 29th, 1891.—No. 297.

Temperature of room 74·7° F. Sickly Mahomedan male, Peer Mahomed, ætat. 40. Chloroformed at the Afzulgunj Hospital at 8h. 41m. 20s. by Miss E. A. Lawrie. Full anæsthesia at 8h. 45m. 25s. Continued until 8h. 46m. 30s.

*Observations.*

H. M. S.

A. 8 41 20.—Chloroform on cap ; blowing regularly.

B. 8 42 0.—Regular breathing ; twenty-eight times in a minute.

C. 8 42 40.—Holding breath ; cap entirely removed.

D. 8 42 50.—Gasped fresh air ; cap brought close again.

E. 8 43 0.—Irregular breathing.

F. 8 43 30.—While more chloroform was being poured into the cap, took three breaths of pure air.

G. 8 43 50.—Irregular breathing ; one breath of pure air.

H. 8 44 30.—Regular breathing ; 26 respirations in a minute.

I. 8 44 50.—Struggling ; one breath of pure air.

J. 8 45 5.—Noisy expiration.

K. 8 45 25.—Stertorous breathing ; stopped chloroform.

L. 8 45 28.—Cornea insensitive. Administration continued afterwards until 8h. 46m. 30s.

## August 29th, 1891.—No. 298.

Temperature of room 74·7° F. Delicate Hindoo child, Nagamah, ætat. 6. Disease—abscess. Operation—incision. Chloroformed at Afzulgunj Hospital at 8h. 52m. 10s. by Miss E. A. Lawrie. Full anæsthesia at 8h. 53m. 5s. Continued until 8h. 59m. 30s.

*Observations.*

H. M. S.

A. 8 52 10.—Chloroform on cap ; breathing regular.

B. 8 52 45.—Cap close ; regular respirations, 28 in a minute.

C. 8 53 5.—Cornea insensitive. Chloroform stopped, and afterwards continued until 8h. 59m. 30s.

## August 29th, 1891.—No. 299.

Temperature of room 74·7° F. Unhealthy Hindoo male, Jumnadas, ætat. 30. Disease—abscess. Operation—incision. Chloroformed at the Afzulgunj Hospital by Surgeon-Major Lawrie at 9h. 33m. 5s. Anæsthesia complete at 9h. 35m. 45s., and continued until 9h. 37m. 30s.

### *Observations.*

| | H. | M. | S. | |
|---|---|---|---|---|
| A. | 9 | 33 | 5. | —Chloroform on cap; regular respirations, 36 a minute. |
| B. | 9 | 33 | 15. | —Cap brought close to face. Regular breathing. |
| C. | 9 | 33 | 30. | —Noisy expiration. |
| D. | 9 | 34 | 5. | —Talking. |
| E. | 9 | 34 | 25. | —While more chloroform was being poured on to the cap two breaths of pure air were taken. |
| F. | 9 | 35 | 5. | —Violent struggling; regular breathing. |
| G. | 9 | 35 | 30. | —Noisy expiration. |
| H. | 9 | 35 | 45. | —Cornea insensitive. Stopped chloroform. Chloroform afterwards administered until 9h. 37m. 30s. |

Chloroform was administered in Case No. 299 by the surgeon. The students were becoming timid, in consequence, it was supposed, of the weekly reports of deaths under anæsthetics in the English medical journals; and were taking from six to eight minutes to produce anæsthesia instead of from three to five. In Case No. 299, full anæsthesia was produced, with regular breathing throughout the administration, in two minutes and forty seconds. Dr. Lawrie afterwards said, "I never wish you to give chloroform against time, but, provided the respiration is normal, there is nothing to be gained by slow anæsthesia. If the breathing is regular you need not be anxious about the patient, as no danger can arise before anæsthesia is complete, whether the chloroform is administered slowly or quickly. Until the stage of complete narcosis is reached therefore your anxiety should be to *make* the patient breathe regularly while he inhales the anæsthetic, and you should not allow him to inhale any chloroform when the breathing is irregular. Beyond this you need have no anxieties whatever."

### August 29th, 1891.—No. 300.

Temperature of room 80·5° F. Sickly Hindoo female, ætat. 32. Disease—abscess. Operation—incision. Chloroformed at the Afzulgunj Hospital by student Miss Williams at 9h. 47m. 30s. Anæsthesia complete at 9h. 52m. 15s. Continued until the operation was finished at 9h. 54m. 15s.

*Observations.*

H. M. S.

A. 9 47 30.—Chloroform on cap ; respirations regular, 17 per minute.

B. 9 48 25.—Regular breathing, 24 in a minute.

C. 9 49 50.—Moving hands.

D. 9 50 10.—Talking.

E. 9 50 15.—Irregular breathing ; one breath of pure air.

F. 9 50 50.—While more chloroform was being poured on to the cap, took two breaths of pure air.

G. 9 52 15.—Cornea insensitive. Cap was afterwards re-applied until 9h. 54m. 15s. when the operation was finished.

### August 29th, 1891.—No. 301.

Temperature of room 80·5° F. Weakly Hindoo male, Parwaty Ram, ætat. 30. Disease—abscess. Operation—incision. Chloroformed at the Afzulgunj Hospital by student Krishtiah at 10h. 4m. 50s. Anæsthesia complete at 10h. 8m. 50s. Continued until 11h. 20m. 0s.

*Observations.*

H. M. S.

A. 10 4 50.—Chloroform on cap.

B. 10 5 15.—Very rapid respirations, 52 a minute.

C. 10 5 30.—Noisy expiration.

D. 10 6 50.—Breathing regular, 24 in a minute.

E. 10 7 30.—Struggling ; one breath of pure air.

F. 10 8 30.—Talking.

G. 10 8 50.—Cornea insensitive. Chloroform stopped. Continued afterwards until the operation was finished at 10h. 20m. 0s.

## August 31st, 1891.—No. 302.

Temperature of room 75·8° F. Healthy Mahomedan male, Mahomed Karimoolla, ætat. 22. Disease—Fibroid tumour of scrotum. Operation—excision. Chloroformed at the Afzulgunj Hospital by Shivram Balkristna (his first administration) at 7h. 51m. 45s. Full anæsthesia at 7h. 57m. 53s; continued until 8h. 8m. 45s.

### *Observations.*

H. M. S.

A. 7 51 45.—Chloroform on cap; blowing hard, 40 times a minute.

B. 7 52 10.—Cap brought close to face.

C. 7 52 20.—Apnœa; cap entirely removed.

D. 7 52 35.—One breath of pure air. Cap re-applied.

E. 7 52 55.—Regular respirations, 20 a minute.

F. 7 53 25.—While more chloroform was being poured on to the cap, took three breaths of pure air.

G. 7 53 55.—Regular respirations, 24 a minute.

H. 7 55 0.—Moaning.

I. 7 55 55.—While more chloroform was being poured on to the cap, took four breaths of pure air.

J. 7 56 30.—Snoring.

K. 7 57 50.—While more chloroform was being poured on to the cap, took two breaths of pure air.

The cap was not re-applied till later as, while the chloroform was being renewed, and before any more was inhaled, the cornea became insensitive and the breathing stertorous.

## August 31st, 1891.—No. 303.

Temperature of room 75·8° F. Thin Mahomedan male child, Fakeer Mahomed, ætat. 5. Disease—stone in the bladder. Operation—lateral lithotomy. Chloroformed at the Afzulgunj Hospital at 8h. 31m. 15s., by student Shivram Balkristna. Full anæsthesia at 8h. 33m. 45s. Continued until 8h. 44m. 30s.

*Observations.*

H. M. S.
A. 8 31 15.—Chloroform on cap; regular crying respirations, 36 a minute.
B. 8 31 50.—Still crying; breathing irregular. One breath of pure air.
C. 8 31 55.—Cap brought close to face; respirations 32, regular.
D. 8 32 50.—While more chloroform was being poured on to the cap, took five breaths of pure air.
E. 8 33 0.—Regular breathing.
F. 8 33 45.—Cornea insensitive; stop chloroform. The administration was afterwards continued until the dressing was completed at 8h. 44m. 30s.

August 31st, 1891.—No. 304.

Temperature of room 75·8° F. Emaciated Mahomedan male, Peer Mahomed, ætat. 46. Disease—stricture of the urethra. Operation—Holt's dilatation of the stricture. Chloroformed at the Afzulgunj Hospital at 8h. 50m. 45s. by student Shivram Balkristna. Full anæsthesia at 8h. 54m. 30s., continued till 8h. 55m. 30s.

*Observations.*

H. M. S.
A. 8 50 45.—Chloroform on cap; rapid blowing, 44 times a minute.
B. 8 51 10.—Cap brought close to face.
C. 8 52 15.—Regular respirations, 20 a minute.
D. 8 53 5.—Moving arms and legs.
E. 8 53 25.—While more chloroform was being poured on to the cap, took three breaths of pure air.
F. 8 53 55.—Irregular breathing; one breath of pure air.
G. 8 54 20.—Noisy expiration.
H. 8 54 30.—Cornea insensitive. Stopped chloroform.

August 31st, 1891.—No. 305.

Temperature of room 81·7° F. Sickly Hindoo male, ætat. 48. Disease—abscess. Operation—incision. Chloroformed at the Afzulgunj Hospital at 9h. 4m. 10s. by Miss E. A. Lawrie. Full anæsthesia at 9h. 7m. 30s. Continued until 9h. 10m. 30s.

*Observations.*

| | H. | M. | S. |
|---|---|---|---|
| A. | 9 | 4 | 10.—Chloroform on cap ; breathing regular, 32 times a minute. |
| B. | 9 | 4 | 55.—Cap brought close to face. |
| C. | 9 | 4 | 55.—Struggling ; one breath of pure air. |
| D. | 9 | 5 | 50.— Do. do. do. |
| E. | 9 | 6 | 10.—Regular respirations, 20 in a minute. |
| F. | 9 | 6 | 45.—While more chloroform was added to the cap, took four breaths of pure air. |
| G. | 9 | 7 | 0.—Regular respirations, 24 a minute. |
| H. | 9 | 7 | 20.—Noisy expiration and moaning. |
| I. | 9 | 7 | 30.—Cornea insensitive ; chloroform stopped, and afterwards continued until the dressings were applied. |

August 31st, 1891.—No. 306.

Temperature of room 81·7° F. Sickly Mahomedan male, Mahomed Ismael, ætat. 51. Disease—cellulitis. Operation—incisions. Chloroformed at the Afzulgunj Hospital at 9h. 17m. 30s. by Miss E. A. Lawrie. Full anæsthesia at 9h. 23m. 0s ; continued till 9h. 27m. 30s.

*Observations.*

| | H. | M. | S. |
|---|---|---|---|
| A. | 9 | 17 | 30.—Chloroform on cap ; rapid regular breathing, 40 in a minute. |
| B. | 9 | 19 | 50.—Regular breathing, 20 in a minute. |
| C. | 9 | 20 | 5.—Coughing. |
| D. | 9 | 20 | 40.—While more chloroform was being added to the cap, took four breaths of pure air. |
| E. | 9 | 21 | 10.—Noisy expiration. |
| F. | 9 | 21 | 25.—Violent struggling. Regular breathing. |
| G. | 9 | 21 | 45.—One breath of pure air. |
| H. | 9 | 22 | 15.—Struggling ; regular breathing. |
| I. | 9 | 22 | 45.—Snoring. |
| J. | 9 | 23 | 0.—Cornea insensitive. Stopped chloroform. Continued afterwards until 9h. 27m. 30s. |

## September, 1st. 1891—No. 307.

Temperature of room 76·5° F. Sickly Hindoo male, Boochaya, ætat. 32. Disease—granular lids. Operation—division of the tarsal cartilages. Chloroformed at the Afzulgunj Hospital at 7h. 53m. 50s. by Miss E. A. Lawrie. Full anæsthesia at 7h. 59m. 25s., kept up until 8h. 1m. 5s. without more chloroform.

### *Observations.*

| | H. | M. | S. | |
|---|---|---|---|---|
| A. | 7 | 53 | 50. | —Chloroform on cap ; blowing vigorously, 28 times a minute. |
| B. | 7 | 54 | 30. | —Apnœa ; cap entirely removed. |
| C. | 7 | 54 | 45. | —One breath of pure air ; cap re-applied. |
| D. | 7 | 55 | 0. | —Regular slow respirations, 16 a minute. |
| E. | 7 | 57 | 15. | —Took two breaths of pure air while more chloroform was being poured into the cap. |
| F. | 7 | 57 | 45. | —Holding breath ; cap entirely removed. |
| G. | 7 | 58 | 0. | —Gasped pure air ; breathing regular. Cap re-applied. |
| H. | 7 | 58 | 55. | —Noisy expiration. |
| I. | 7 | 59 | 25. | —Cornea insensitive. Stopped chloroform. |

## September 1st, 1891.—No. 308.

Temperature of room 76·6° F. Strong Hindoo male, Ramanna, ætat. 25. Disease—suppurating bubo. Operation—excision of inguinal glands. Chloroformed at the Afzulgunj Hospital at 8h. 17m. 40s. by student Shivram Balkristna. Full anæsthesia at 8h. 23m. 20s. Anæsthesia maintained until 8h. 26m. 15s.

### *Observations.*

| | H. | M. | S. | |
|---|---|---|---|---|
| A. | 8 | 17 | 40. | —Chloroform on cap ; blowing very rapidly, 52 times a minute. |
| B. | 8 | 18 | 15. | —Blowing 68 times a minute. |
| C. | 8 | 18 | 40. | —Apnœa ; cap entirely removed. |
| D. | 8 | 18 | 50. | —One breath of pure air, followed by regular breathing, 20 times a minute. Cap re-applied. |

| | H. | M. | S. |
|---|---|---|---|
| E. | 8 | 19 | 30.—Apnœa ; cap entirely removed until 8h. 20m. 20s. (50 seconds.) |
| F. | 8 | 20 | 20.—One breath of pure air ; cap re-applied. |
| G. | 8 | 21 | 0.—Noisy expiration. |
| H. | 8 | 21 | 25.—Two breaths of pure air while adding more chloroform. |
| I. | 8 | 22 | 40.—Noisy expiration. |
| J. | 8 | 23 | 20.—Cornea insensitive. Anæsthesia maintained by giving more chloroform from time to time until 8h. 26m. 15s. |

September 1st, 1891.—No. 309.

Temperature of room 76·6° F. Old worn-out feverish Mahomedan male, Dawod Khan, ætat. 81. Disease—cat-bite. Temperature in mouth 102·7° F. Operation—excision. Chloroformed at the Afzulgunj Hospital at 8h. 32m. 20s. by Miss Lawrie. Fully anæsthetised at 8h. 36m. 40s.

*Observations.*

| | H. | M. | S. |
|---|---|---|---|
| A. | 8 | 32 | 20.—Chloroform on cap ; rapid blowing, 52 times a minute |
| B. | 8 | 33 | 5.—Cap brought close to face ; regular breathing, 32 a minute. |
| C. | 8 | 33 | 20.—Apnœa ; cap entirely removed. |
| D. | 8 | 33 | 30.—One breath of pure air ; cap re-applied. |
| E. | 8 | 34 | 0.—Struggling ; breathing regular. |
| F. | 8 | 34 | 40.—Holding breath ; cap entirely removed. |
| G. | 8 | 34 | 55.—Gasped pure air ; regular breathing. Cap re-applied. |
| H. | 8 | 35 | 15.—Struggling ; regular breathing. |
| I. | 8 | 36 | 0.—Noisy expiration ; 36 respirations a minute. |
| J. | 8 | 36 | 40.—Cornea insensitive ; stopped chloroform. |
| K. | 8 | 37 | 5.—Stertorous breathing ; no more chloroform had been administered from 8h. 36m. 40s. (*Vide* remarks after Case No. 278.) |

## September 1st, 1891.—No. 310.

Temperature of room 76·6° F. Worn out old Mahomedan Kurreemudin, ætat. 71. Disease—senile hypertrophy of prostate. Operation—perineal section. Chloroformed at the Afzulgunj Hospital at 8h. 48m. 50s. by Shivram Balkristna. Full anæsthesia at 8h. 51m. 45s. Continued until 8h. 56m. 50s.

### *Observations.*

| | H. | M. | S. |
|---|---|---|---|
| A. | 8 | 48 | 50.—Chloroform on cap ; blowing gently 38 times a minute. |
| B. | 8 | 49 | 30.—Cap brought close to the face ; regular breathing 36 per minute. |
| C. | 8 | 49 | 50.—Coughing. |
| D. | 8 | 50 | 10.—Regular respirations ; 28 a minute. |
| E. | 8 | 50 | 25.—Talking. |
| F. | 8 | 50 | 35.—Struggling ; regular breathing. |
| G. | 8 | 51 | 10.—Noisy expiration ; 32 respirations a minute. |
| H. | 8 | 51 | 45.—Cornea insensitive ; stopped chloroform. Continued afterwards until the operation was finished at 8h. 56m. 50s. |

## September 1st, 1891.—No. 311.

Temperature of room 83·5° F. Sickly Mahomedan male, Sayyed Mohidin, ætat. 45. Disease—fistula in ano. Operation—incision. Chloroformed at the Afzulgunj Hospital at 9h. 52m. 50s. by student Shivram Balkristna. Full anæsthesia at 9h: 56m. 45s. Continued until 10h. 1m. 5s.

### *Observations.*

| | H. | M. | S. |
|---|---|---|---|
| A. | 9 | 52 | 50.—Chloroform on cap ; blowing very rapidly, 60 times a minute. |
| B. | 9 | 53 | 20.—Apnœa ; cap entirely removed. |
| C. | 9 | 53 | 35.—Took one breath of pure air ; cap re-applied. |
| D. | 9 | 53 | 45.—Regular respirations, 24 a minute. |

| | H. | M. | S. |
|---|---|---|---|
| E. | 9 | 54 | 20.—While more chloroform was added to cap, took three breaths of pure air. |
| F. | 9 | 54 | 40.—Coughing ; one breath of pure air. |
| G. | 9 | 55 | 10.— Do. do. do. |
| H. | 9 | 55 | 30.—Noisy expiration. |
| I. | 9 | 55 | 50.—Holding breath ; cap entirely removed. |
| J. | 9 | 56 | 15.—Gasped in pure air; cap re-applied ; regular breathing. |
| K. | 9 | 56 | 25.—Moaning. |
| L. | 9 | 56 | 45.—Stertorous breathing ; cornea at same time insensitive. Stopped chloroform ; afterwards continued until 10h. 1m. 5s. |

September 2nd, 1891.—No. 312.

Temperature of room 72·9° F. Delicate Hindoo child, Baloo, ætat. 4. Disease—phymosis. Operation—circumcision. Chloroformed at the Afzulgunj Hospital at 7h. 22m. 35s. by student Miss Williams. Full anæsthesia at 7h. 24m. 0s., administration ended at 7h. 28m. 30s.

*Observations.*

| | H. | M. | S. |
|---|---|---|---|
| A. | 7 | 22 | 35.—Chloroform on cap ; crying. |
| B. | 7 | 23 | 25.—Regular respirations, 24 a minute. |
| C. | 7 | 24 | 0.—Cornea insensitive ; stopped chloroform ; anæsthesia afterwards kept up in the usual way until the operation was finished at 7h. 28m. 30s. |

September 2nd, 1891.—No. 313.

Temperature of room 73·0° F. Healthy young Mahomedan female, Abbass Bee, ætat. 20. Disease—cystic tumour. Operation—excision. Chloroformed at the Afzulgunj Hospital at 8h. 0m. 45s. by Miss E A. Lawrie. Full anæsthesia at 8h. 6m. 20s. Anæsthesia maintained until the operation was finished at 8h. 15m. 30s.

*Observations.*

| | H. | M. | S. |
|---|---|---|---|
| A. | 8 | 0 | 45.—Chloroform on cap ; regular gentle blowing, 24 times a minute. |
| B. | 8 | 1 | 30.—Cap close to face. |
| C. | 8 | 1 | 40.—Talking ; breathing regular. |
| D. | 8 | 2 | 45.—Regular respirations, 20 a minute. |
| E. | 8 | 3 | 10.—Took three breaths of pure air while more chloroform was being added to the cap. |
| F. | 8 | 4 | 0.—Regular respirations, 20 a minute. |
| G. | 8 | 4 | 55.—Snoring ; took two breaths of pure air while more chloroform was being added to the cap. |
| H. | 8 | 5 | 10.—Moaning. |
| I. | 8 | 5 | 45.—Snoring. |
| J. | 8 | 6 | 20.—Cornea insensitive ; stopped chloroform. Anæsthesia was subsequently kept up in the usual way until 8h. 15m. 30s. |

September 2nd, 1891.—No. 314.

Temperature of room 77·4° F. Sickly old Hindoo, Syanna, ætat. 75. Disease—panophthalmitis. Operation—excision of the eye-ball. Chloroformed at the Afzulgunj Hospital at 8h. 44m. 5s. by Miss E. A. Lawrie. Full anæsthesia at 8h. 48m. 50s. Continued until 8h. 55m. 15s.

*Observations.*

| | H. | M. | S. |
|---|---|---|---|
| A. | 8 | 44 | 5.—Chloroform on cap ; blowing 32 times a minute. |
| B. | 8 | 44 | 35.—Cap close to face ; regular breathing. |
| C. | 8 | 45 | 55.—More chloroform ; three breaths of pure air. |
| D. | 8 | 46 | 45.—Moving arms. |
| E. | 8 | 47 | 15.—More chloroform ; two breaths of pure air. |
| F. | 8 | 47 | 20.—Struggling ; regular breathing. |
| G. | 8 | 48 | 5.—Noisy expiration. |
| H. | 8 | 48 | 30.—Snoring. |
| I. | 8 | 48 | 50.—Cornea insensitive ; stopped chloroform ; anæsthesia afterwards kept up in the usual way until the operation was finished at 8h. 55m. 15s. |

## September 2nd, 1891.—No. 315.

Temperature of room 77·8° F. Strong healthy male, Kishen Singh, ætat. 28. Disease—necrosis of lower jaw. Operation—removal of dead bone. Chloroformed at the Afzulgunj Hospital at 9h. 3m. 40s Full anæsthesia at 9h. 10m. 10s.

### *Observations.*

H. M. S.

A. 9 3 40.—Chloroform on cap ; rapid blowing, 54 a minute.

B. 9 4 25.—Cap brought close to face ; respirations regular, 28 a minute.

C. 9 5 30.—Regular breathing, 24 a minute.

D. 9 6 0.—Took three breaths of pure air while more chloroform was poured into the cap.

E. 9 6 45.—Coughing.

F. 9 7 15.—Moaning.

G. 9 10 10.—Cornea insensitive ; stopped chloroform.

## September 3rd, 1891.—No. 316.

Temperature of room 73·6° F. Healthy Mahomedan male, Syed Abdul Wahab, ætat. 32. Disease—stricture of the urethra and retention of urine. Operation—catheterism. Chloroformed at the Afzulgunj Hospital at 7h. 56m. 40s. by Shivram Balkristna. Full anæsthesia at 8h. 0m. 20s. ; continued until 8h. 2m. 30s.

### *Observations.*

H. M. S.

A. 7 56 40.—Chloroform on cap ; blowing rapidly 40 times a minute.

B. 7 57 0.—Cap close to face ; regular breathing 32 a minute.

C. 7 57 40.—Holding breath ; stopped chloroform ; cap entirely removed.

D. 7 57 58.—Gasped pure air ; chloroform again.

E. 7 58 15.—Regular respirations, 20 a minute.

F. 7 58 40.—Took two breaths of pure air while chloroform was being poured into cap.

H. M. S.
G. 7 58 55.—Irregular breathing ; one breath of pure air.
H. 7 59 15.—Regular breathing, slow and moaning, 16 a minute.
I. 7 59 40.—Prolonged moaning expirations.
J. 8 0 20.—Cornea insensitive ; chloroform stopped. Immediately afterwards the chest was heaving, but no air was entering. The jaw was pushed forward, and the breathing became at once normal.

### September 3rd, 1891.—No. 317.

Temperature of room 74·5° F. Sickly Mahomedan male, Nanna Sahib, ætat. 64. Disease—suppurating hydrocele. Temperature in rectum 103·4° F. before the operation. Operation—excision of part of sac. Chloroformed at the Afzulgunj Hospital at 8h. 44m. 30s. by Shivram Balkristna. Full anæsthesia at 8h. 48m. 30s.; continued until 8h. 51m. 15s.

### *Observations.*

H. M. S.
A. 8 44 30.—Chloroform on cap ; rapid noisy blowing, 56 times a minute.
B. 8 45 20.—Cap close to face.
C. 8 45 40.—Apnœa ; cap removed.
D. 8 45 55.—One breath of pure air. Chloroform again.
E. 8 46 15.—Regular respirations, 20 a minute.
F. 8 46 50.—Took three breaths of pure air while more chloroform was added to the cap.
G. 8 47 20.—Moving arms and legs ; talking incoherently.
H. 8 47 45.—Struggling ; one breath of pure air.
I. 8 48 20.—Spluttering and snoring.
J. 8 48 30.—Cornea insensitive; stopped chloroform.
K. 8 48 50.—Stertorous breathing; jaw pushed forward ; normal respiration.
L. 8 51 15.—Operation finished.

September 3rd, 1891.—No. 318.

Temperature of room 74·5° F. Very sickly Hindoo male, Kristna, ætat. 25. Disease—spreading gangrene. Operation—exploration. Chloroformed at the Afzulgunj Hospital at 9h. 24m. 50s. by Shivram Balkristna. Full anæsthesia at 9h. 30m. 5s.

*Observations.*

H. M. S.

A. 9 24 50.—Chloroform on cap ; blowing 40 times a minute.

B. 9 25 20.—Cap close to face ; coughing.

C. 9 26 10.—Took one breath of pure air while more chloroform was added to the cap.

D. 9 26 45.—Struggling ; one breath of pure air.

E. 9 27 10.—Regular respirations, 28 a minute.

F. 9 28 55.—Took three breaths of pure air while more chloroform was poured into the cap.

G. 9 29 10.—Snoring.

H. 9 30 5.—Cornea insensitive ; stopped chloroform.

September 3rd, 1891.—No. 319.

Temperature of room 74·5° F. Old decrepit Mahomedan, Sayyad Ashraf, ætat. 82. Disease—senile hypertrophy of the prostate. Operation—perineal section. Chloroformed at the Residency Hospital at 11h. 30m. 15s. by student Ismail Khan. Full anæsthesia at 11h. 33m. 50s. ; continued until the operation was finished at 11h. 37m. 15s.

*Observations.*

H. M. S.

A. 11 30 15.—Chloroform on cap ; blowing gently 36 times a minute.

B. 11 30 45.—Cap close to face.

C. 11 31 5.—Regular breathing, 28 times a minute.

D. 11 31 30.— Do. do. 20 do. do.

E. 11 31 50.—Took two breaths of pure air while more chloroform was added to the cap.

H. M. S.

F. 11 32 45.—Holding breath ; cap entirely removed.

G. 11 33 15.—Gasped pure air ; chloroform again.

H. 11 33 40.—Irregular breathing ; one breath of pure air.

I. 11 33 50.—Snoring ; cornea insensitive ; stopped chloroform. Continued afterwards till the operation was finished at 11h. 37m. 15s.

September 4th, 1891.—No. 320.

Temperature of room 72·9° F. Weakly emaciated Hindoo male, Lutchannah, ætat. 31. Disease—liver abscess. Operation—aspiration. Chloroformed at he Afzulgunj Hospital at 7h. 5m. 25s, by Shivram Balkristna. Full anæsthesia t 7h. 9m. 30s. ; continued until 7h. 12m. 30s.

*Observations.*

H. M. S.

A. 7 5 25.—Chloroform on cap ; blowing rapidly, 50 times a minute.

B. 7 6 15.—Cap close to face ; regular respirations, 44 a minute.

C. 7 7 45.—Regular respirations, 20 a minute.

D. 7 8 25.—Took three breaths of pure air while more chloroform was added to the cap.

E. 7 9 15.—Noisy expiration.

F. 7 9 30.—Cornea insensitive ; stopped chloroform ; continued afterwards until 7h. 12m. 30s.

September 4th, 1891.—No. 321.

Temperature of room 72·9° F. Weakly Hindoo male infant, Gunguloo, ætat. two months. Disease—abscess. Operation—incision. Chloroformed at the Afzulgunj Hospital at 8h. 34m. 45s. by Miss Williams. Full anæsthesia at 8h. 36m. 5s. Continued until 8h. 39m. 30s.

*Observations.*

H. M. S.

A. 8 34 45.—Chloroform on cap ; crying and coughing.

B. 8 35 15.—Regular respirations, 48 a minute.

C. 8 36 25.—Cornea insensitive ; stopped chloroform. Continued afterwards until 8h. 39m. 30s.

### September 5 th, 1891.—No. 322.

Temperature of room, 74·5° F. Sickly Hindoo male, Sayboo, ætat. 25. Disease—bubo. Operation—excision of the inguinal glands. Chloroformed at the Afzulgunj Hospital at 8h. 32m. 10s. by Shivram Balkristna. Full anæsthesia at 8h. 36m. 35s. Continued until 8h. 42m. 30s.

*Observations.*

H. M. S.
A. 8 32 10.—Chloroform on cap; blowing gently, 44 times a minute.
B. 8 32 50.—Cap close to face ; regular respirations, 20 a minute.
C. 8 33 20.—Irregular breathing, one breath of pure air.
D. 8 33 30.—Regular respirations, 24 a minute.
E. 8 34 30.—Took two breaths of pure air while more chloroform was added to the cap.
F. 8 35 0.—Struggling, one breath of pure air.
G. 8 35 15.—Regular breathing, 28 times a minute.
H. 8 35 55.—Snoring.
I. 8 36 20.—Spluttering.
J. 8 36 35.—Cornea insensitive and breathing stertorous simultaneously ; stopped chloroform ; anæsthesia afterwards kept up till 8h. 42m 30s.

### September 5th, 1891.—No. 323.

Temperature of room 74·5° F. Weak Hindoo child, Nursoo, ætat. 2. Disease—abscess. Operation—incision. Chloroformed at the Afzulgunj Hospital at 8h. 45m. 55s. by Miss Williams. Full anæsthesia at 8h. 47m. 50s. Continued until 8h. 51m. 30s.

*Observations.*

H. M. S.
A. 8 45 55.—Chloroform on cap ; crying.
B. 8 46 30.—Cap close ; regular respirations, 27 a minute.
C. 8 47 40.—Took four breaths of pure air while more chloroform was added.
D. 8 47 50.—*Cornea insensitive ; stopped chloroform ;* continued afterwards until the dressings were applied at 8h. 51m. 30s.

## September 6th, 1891.—No. 324.

**Temperature of room 77·4° F. Very old Hindoo female, Kundee, ætat. 84. Injury—compound fracture of the right leg. Operation—amputation below the knee. Chloroformed at the Afzulgunj Hospital at 9h. 45m. 55s. by Miss E. A. Lawrie. Full anæsthesia at 9h. 48m. 10s. Continued until the operation was finished at 10h. 25m. 0s.**

### *Observations.*

| | H. | M. | S. | |
|---|---|---|---|---|
| A. | 9 | 45 | 55. | —Chloroform on cap; breathing regularly and talking. |
| B. | 9 | 46 | 5. | —Struggling ; regular breathing. |
| C. | 9 | 46 | 25. | —Cap close to face ; regular respirations, 20 a minute. |
| D. | 9 | 47 | 0. | —Irregular respiration and struggling ; one breath of pure air ; regular breathing. |
| E. | 9 | 47 | 15. | —Took three breaths of pure air while chloroform was being poured on to cap. |
| F. | 9 | 47 | 30. | —Spitting. |
| G. | 9 | 47 | 35. | —Regular breathing, 24 a minute. |
| H. | 9 | 48 | 5. | —Cornea insensitive ; stopped chloroform. Anæsthesia afterwards maintained in the usual way till the operation was finished and the dressings applied at 10h. 25m. 0s. |

## September 7th, 1891.—No. 325.

Temperature of room 74·9° F. Strong, muscular Mahomedan male, excessively excited and nervous, Rusool Mahomed, ætat. 28. Disease epithelioma of skin of back. Operation—excision. Chloroformed at the Afzulgunj Hospital at 8h. 52m. 35s. by Shivram Balkristna. Full anæsthesia at 8h. 57m. 0s. *Continued until the operation was finished at 9h. 15m. 30s. Vomited* a small quantity of bile at 9h. 16m. 10s.

*Observations.*

H. M. S.

A. 8 52 35.—Chloroform on cap; blowing 42 a minute.
B. 8 53 10.—Cap close to face.
C. 8 53 50.—Struggle; one breath of pure air.
D. 8 54 40.—Violent struggling; one breath of pure air.
E. 8 54 55.—Screaming; breathing regular.
F. 8 55 5.—One breath of pure air.
G. 8 56 0.—Took two breaths of pure air while more chloroform was added to the cap.
H. 8 56 45.—Snoring loudly.
I. 8 57 0.—Cornea insensitive; stopped chloroform.
J. 8 57 35.—Breathing stertorous; jaw pushed forward, breathing regular at once.

This case is another example of anæsthesia deepening after discontinuance of the inhalation of chloroform. This appears to occur in patients who are excited and in whom it is difficult to make the respiration quite regular. It is in patients of this kind that over-dosing is so liable to take place; and the explanation of what happens is to be found in Experiment No. 64.

September 7th, 1891.—No. 326.

Temperature of room 77·7° F. Weak sickly anæmic Hindoo male, Bala Prasad, ætat. 28. Disease—strangulated inguinal hernia. Operation—herniotomy and the radical cure. Chloroformed at the Afzulgunj Hospital at 9h. 23m. 40s. by Shivram Balkristna. Full anæsthesia at 9h. 28m. 15s.; continued until the operation was finished at 10h. 0m. 45s.

*Observations.*

H. M. S.

A. 9 23 40.—Chloroform on cap; blowing rapidly, 56 times a minute.
B. 9 24 5.—Cap close to face; regular respirations, 32 a minute.
C. 9 25 30.—Regular respirations, 28 a minute.
D. 9 25 50.—Short struggle; one breath of pure air.
E. 9 27 35.—Took two breaths of pure air while more chloroform was added.

| | H. | M. | S. | |
|---|---|---|---|---|
| F. | 9 | 27 | 45.— | Talking incoherently. |
| G. | 9 | 28 | 15.— | Cornea insensitive; stopped chloroform. Anæsthesia kept up, afterwards until the operation was finished at 10h. 0m. 45s. |

As a clinical record of facts the above cases are no less valuable in their way than the record of the facts observed in the experiments of the Hyderabad Commission, of which they form the counterpart. Clinical experience confirms the experimental data of the Commission, and proves that the first effect of chloroform inhalation with regular breathing is normal narcosis, so called because it is free from risk. Accordingly the first step in chloroform administration should be to secure inhalation with regular breathing, and the Commission's experiments as well as the clinical cases co-incide in showing that this is the main difficulty the chloroformist has to contend with. Both prove that apart from mere resistance, any idiosyncrasies that may display themselves, whether in man or animals, before the stage of full narcosis is reached, are not due to the direct effects of chloroform, but are caused by the peculiarities in, or the distinctive features of the respiration in the subject operated upon. For example, the distinctive features of the respiratory function which enable rabbits to burrow in sand, and goats to career across rocky heights, lend themselves very readily to the irregular respiration, holding the breath, and prolonged reflex stoppage of the heart, which make it by no means easy to obtain regular blood-pressure tracings with these animals in the first stage of chloroform administration.[*] Similar effects may be produced clinically in patients under chloroform by peculiarities of the respiration depending upon temperament and disease.[†] In the beginning of chloroform inhalation fear or excitement or certain phases of disease may give rise to irregular respiration, which, though harmless in itself, may lead to irregular inhalation and overdosing. It is therefore incumbent on the chloroformist never to allow the patient to inhale chloroform when the breathing is irregular or abnormal. In this way normal anæsthesia can be ensured, and accidents in the early stages of chloroform narcosis, which are said to be unavoidable and due to chloroform syncope, can be prevented. When once surgeons admit that normal anæsthesia is all the chloroformist has to produce, chloroform syncope will become a figment of the past. The clinical and experimental data of the Hyderabad Commission completely expose the fallacy underlying the wide-spread belief in the direct action of chloroform upon the heart, a belief which is founded mainly on fatalities under chloroform that have never been proved to be due to anything but abnormal inhalation.

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[*] *Vide* Experiments Nos. 101, 102, 152, 171 and 172 on rabbits; and Nos. 136, 137, 141, 143, 175 and 176 on goats. It is extremely difficult to make any animal respire regularly in the first stage of chloroform inhalation, especially rabbits and goats. This difficulty may be overcome by artificial respiration through a tracheal tube and pumping chloroform into the lungs with bellows: but this unnatural process entirely vitiates the experiment. Natural breathing is no longer possible, and the normal oxygenation of the blood must be interfered with.

[†] *Vide* Clinical cases, Nos. 285, 290, 316 and 325, which show the effect of temperament; and Nos. 309 and 317 which show the effect of disease.

Much discussion has taken place lately with reference to the quality of the chloroform as a factor in anæsthesia. It may therefore be as well to note that in all the second and in more than half the first series of cases reported in this section, the anæsthetic employed was Duncan and Flockhart's methylated chloroform, and that vomiting occurred in less than five per cent. of the administrations.

The Commission does not attempt to make its conclusions retrospective. It is sufficient to recall the fact that deaths with chloroform have, with few exceptions, happened in the practice of those who take the pulse as a guide as to its effects, and to repeat that the pulse can only give indications of actual danger and of abnormal administration. When it is considered how universal has become the custom of taking the pulse as a guide in chloroform inhalation, more especially during the last ten years, it is evident that but for natural safeguards which surround the administration of chloroform, namely, lowering of the blood-pressure, stoppage of the respiration, and stoppage of the heart, the mortality from abnormal inhalation would have been considerably higher than the one in so many hundreds or thousands shown in the useful statistics of Mr. Roger Williams and Professor Wood.

The Commission desires to prove that in the practice of surgery death from chloroform should be impossible. The question is : Will any surgeon henceforth accept the responsibility of refusing to follow the teaching of the Hyderabad Chloroform Commission, seeing that it demonstrates how normal anæsthesia can invariably be secured ?

## PART X.—PRACTICAL CONCLUSIONS.

The following are the practical conclusions which the Commission think may fairly be deduced from the experiments recorded in this report :—

I. The recumbent position on the back and absolute freedom of respiration are essential.

II. If during an operation the recumbent position on the back cannot, from any cause, be maintained during chloroform administration, the utmost attention to the respiration is necessary to prevent asphyxia or an overdose. If there is any doubt whatever about the state of respiration, the patient should be at once restored to the recumbent position on the back.

III. To ensure absolute freedom of respiration, tight clothing of every kind, either on the neck, chest, or abdomen, is to be strictly avoided ; and no assistants or by-standers should be allowed to exert pressure on any part of the patient's thorax or abdomen, even though the patient be struggling violently. If struggling does occur, it is always possible to hold the patient down by pressure on the shoulders, pelvis, or legs without doing anything which can by any possibility interfere with the free movements of respiration.

IV. An apparatus is not essential, and ought not to be used as, being made to fit the face, it must tend to produce a certain amount of asphyxia. Moreover, it is apt to take up part of the attention which is required elsewhere. In short, no matter how it is made, it introduces an element of danger into the administration. A convenient form of inhaler is an open cone or cap with a little absorbent cotton inside at the apex.

V. At the commencement of inhalation care should be taken, by not holding the cap too close over the mouth and nose, to avoid exciting struggling, or holding the breath. If struggling or holding the breath do occur, great care is necessary to avoid an over-dose during the deep inspirations which follow. When quiet breathing is ensured as the patient begins to go over, there is no reason why the inhaler should not be applied close to the face ; and all that is then necessary is to watch the cornea and to see that the respiration is not interfered with.

VI. In children crying ensures free admission of chloroform into the lungs; but as struggling and holding the breath can hardly be avoided, and one or two whiffs of chloroform may be sufficient to produce complete insensibility, they should always be allowed to inhale a little fresh air during the first deep inspirations which follow. In any struggling persons, but especially in children, it is essential to remove the inhaler after the first or second deep inspiration, as enough chloroform may have been inhaled to produce deep anæsthesia, and this may only appear or may deepen after the chloroform is stopped (*vide supra* sub-paragraphs 2 and 9 of conclusions in paragraph 30). Struggling is best avoided in adults by making them blow out hard after each inspiration during the inhalation.

VII. The patient is, as a rule, anæsthetised and ready for the operation to be commenced when unconscious winking is no longer produced by touching the surface of the eye with the tip of the finger. The anæsthetic should never, under any circumstances, be pushed till the respiration stops; but when once the cornea is insensitive, the patient should be kept gently under by occasional inhalations and not be allowed to come out and renew the stage of struggling and resistance.

VIII. As a rule, no operation should be commenced until the patient is fully under the influence of anæsthetic so as to avoid all chance of death from surgical shock or fright.

IX. The administrator should be guided as to the effect entirely by the respiration. His only object, while producing anæsthesia, is to see that the respiration is not interfered with.

X. If possible, the patient's chest and abdomen should be exposed during chloroform inhalation, so that the respiratory movements can be seen by the administrator. If anything interferes with the respiration in any way, however slightly, even if this occurs at the very commencement of the administration; if breath is held, or if there is stertor, the inhalation should be stopped until the breathing is natural again. This may sometimes create delay and inconvenience with inexperienced administrators, but experience will make any administrator so familiar with the respiratory functions under chloroform that he will, in a short time, know almost by intuition whether anything is going wrong, and be able to put it right without delay before any danger arises.

XI. If the breathing becomes embarrassed, the lower jaw should be pulled, or pushed from behind the angles, forward, so that the lower teeth protrude in front of the upper. This raises the epiglottis and frees the larynx.

At the same time, it is well to assist the respiration artificially until the embarrassment passes off.

XII. If by any accident the respiration stops, artificial respiration should be commenced at once, while an assistant lowers the head and draws forward the tongue with catch-forceps, by Howard's method, assisted by compression and relaxation of the thoracic walls. Artificial respiration should be continued until there is no doubt whatever that natural respiration is completely re-established.

XIII. A small dose of morphia may be injected subcutaneously before chloroform inhalation as it helps to keep the patient in a state of anæsthesia in prolonged operations. There is nothing to show that atropine does any good in connection with the administration of chloroform and it may do a very great deal of harm.

XIV. Alcohol may be given with advantage before operations under chloroform, provided it does not cause excitement, and merely has the effect of giving a patient confidence and steadying the circulation.

The Commission has no doubt whatever that, if the above rules be followed, chloroform may be given in any case requiring an operation with perfect ease and absolute safety so as to do good without the risk of evil.

The practical conclusions are reproduced here in their original form. They were written before the Commission had had time to realise the full meaning of their experimental data, or opportunity to put them to the test of clinical experience. They were not regarded as final by the Commission, as it was hoped and expected that surgeons and physiologists would examine the tracings and descriptions of the experiments for themselves, and draw their own conclusions from them.

It only remains for the Commission to show, by comparison of Syme's principles with the facts established that it has accomplished the object for which it was appointed. "The Commission was applied for because it was felt that Syme's principles, which both experience and experiment had shown to be pratically sound, must be founded upon a firm physiological basis." In his original lecture Syme said, "The points that we consider of the greatest practical importance in the administration of Chloroform are"—

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|---|---|
| (1) "First a free admixture of air with the vapour of chloroform, to ensure which a soft porous material is employed * * * we use no apparatus whatever." | (1) The Commission has shown that a free admixture of air with the vapour of chloroform alone ensures normal respiration and normal anæsthesia,[*] and that no form of apparatus or inhaler can determine regular breathing.[†] |
| (2) Syme said, "Secondly, if this [*viz. a free admixture of air with the vapour of chloroform*] be attended to, the more rapidly the chloroform is given the better till the effect is produced ; and hence we do not stint the quantity of the chloroform." | (2) The Commission has demonstrated that there is nothing to be gained by slow anæsthesia, and that the strongest concentration of the vapour of chloroform that is compatible with regular breathing constitutes the safe dilution of the anæsthetic.[‡] This does not mean that the patient is to be forced to inhale over concentrated vapour. Any concentration, or any method of administration whatsoever, which interferes with or stops the patient's respiration must impede the inhalation, and by importing into the administration the risk of asphyxia must produce abnormal anæsthesia. |
| (3) Thirdly, Syme said, "we never continue beyond the point when the patient is fully under the influence of the anæsthetic." | (3) This point is now self-evident. Normal anæsthesia is all the chloroformist has to produce. |
| (4) "Then—and this is a most important point—we are guided as to the effect, not by the circulation but en- | (4) This is the only point on which the Hyderabad Commission has improved upon Syme's teaching. The |

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[*] *Vide* especially Experiments Nos. 148, 168, 169, 170 and Ludwig III and IV of 186.

[†] *Vide* page 298, and experiment No. 41 page 41.

[‡] *Vide* page 397.

tirely by the respiration ; you never see any body here [Edinburgh] with his finger on the pulse while chloroform is given."

pulse can never be a guide to normal chloroform anæsthesia. Syme said that the duty of the chloroformist was *to be guided* by the respiration ; the Commission has proved that it is the chloroformist's duty not to be guided by, but to *guide* the respiration, and to keep the inhalation regular so as always to produce normal anæsthesia.

The report thus ends by showing how thoroughly the Hyderabad Commission has succeeded in proving that the late Professor Syme's principles of chloroform administration are right. This is the most fitting tribute the Commission can pay to the genius and wisdom of the illustrious surgeon, who, by his teaching and example. elevated and ennobled British surgery and paved the way for the new era inaugurated while yet he had full time to appreciate it, by his equally illustrious son-in-law Sir Joseph Lister.

EDWARD LAWRIE, *President.*
T. LAUDER BRUNTON, }
G. BOMFORD, } *Members.*
RUSTOMJI D. HAKIM, }

# PART XI.—PLATES.

## NOTE ON EXPERIMENT No. 169.

Experiment No. 169.—One Ludwig tracing—reading from left to right, and one Fick—reading from right to left. The Ludwig tracing consists of respiratory and pulse curves ; and the pulse curve is also shown from time to time on the Fick tracing. The Ludwig trace shows that after anæsthesia was produced at 11h. 27m. 40s., the respiratory curves became smaller and smaller, and at 11h. 30m., before Fick reading 3, they entirely disappeared. The pulse curve then failed rapidly and, after Fick reading 4, is to be seen as a mere line till the heart stopped. A needle was inserted into the heart after the respiration stopped and moved, vigorously at first and then more and more slowly, until 11h. 34m. 45s.

### *Observations.*

| | H. | M. | S. | |
|---|---|---|---|---|
| A. | 11 | 27 | 5 | Chloroform gently and continuously with regular breathing. |
| B. | 11 | 27 | 40 | Cornea insensitive. |
| C. | 11 | 30 | 55 | Respiration stopped. Needle in heart. |
| D. | 11 | 34 | 45 | Heart stopped. |

Experiment No. 169 shows the effect of uncomplicated chloroform poisoning pushed till death. Anæsthesia was produced at 11h. 27m. 40s. The administration was kept up afterwards for 3m. 15s., during which period the respiratory function was failing, and at 11h. 30m. 55s. all respiratory movements had come to an end. No attempt was made to restore the animal, and it died. It is obvious that the supply of Oxygen to the blood, and through it to the heart, was diminishing, in direct proportion to the deepening of the narcosis of the respiratory centre and the increasing inefficiency of the respiratory function, during the three minutes before the respiratory movements quite ceased. The weakening of the heart, which is perceptible in the pulse-curve of the Ludwig tracing, was, therefore, clearly due to gradual arrest of its nutrition from failure of the respiration ; and it cannot be attributed in any degree to the direct action of chloroform, unless the effect of failure of the respiration on the heart is entirely ignored. After the stoppage of the respiration, the heart's action became more and more enfeebled, until its nutrition was finally arrested owing to the fact that its supply of Oxygen was completely exhausted ; and the heart ceased to beat at 11h. 34m. 45s.

Photo. No. 262. Expt. No. 117. Ludwig tracing II. Reads from left to right. See page 98.

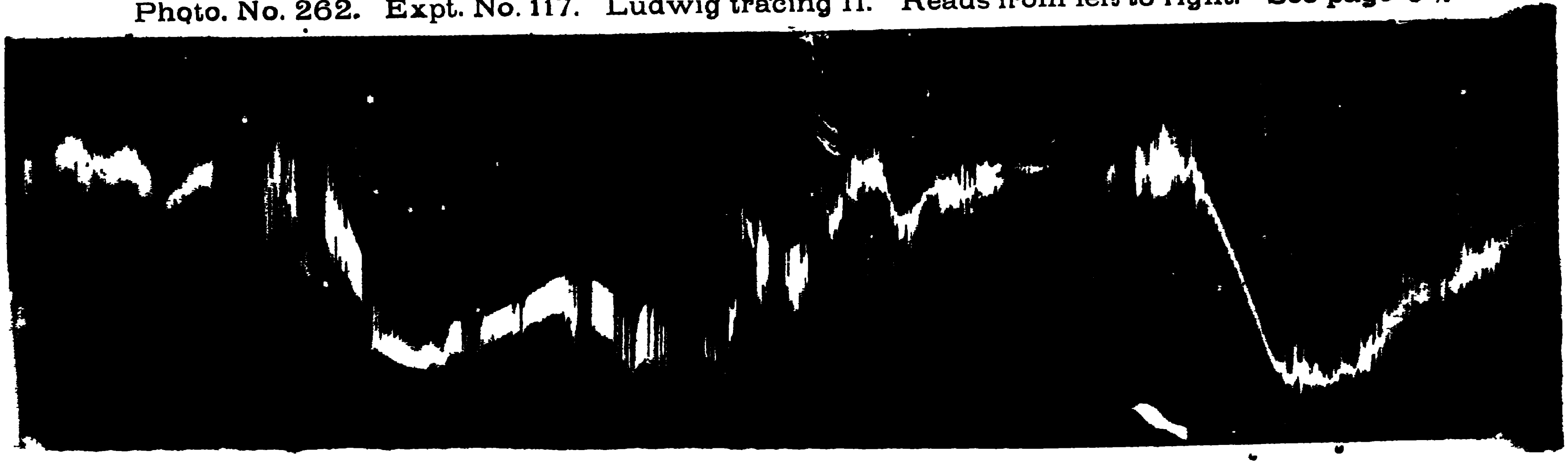
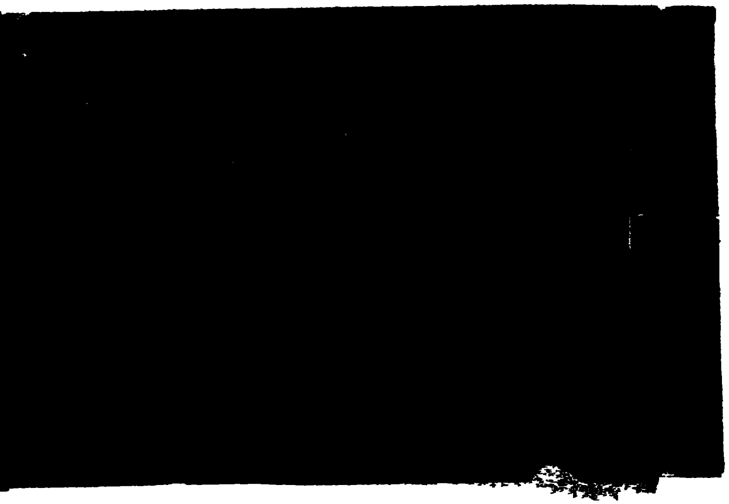


Photo. No. 368. Expt. No. 169. Ludwig tracing.
Reads from left to right.

Fick tracing. Reads from right to left.
See page 130.



*Calcutta Phototype Co.*

Plate I.

Photo. No. 114. Expt. No. 64. See page 53. Fick tracing II. Reads from right to left. Shows cardiac arrest from vagus irritation and chloroform administration combined.

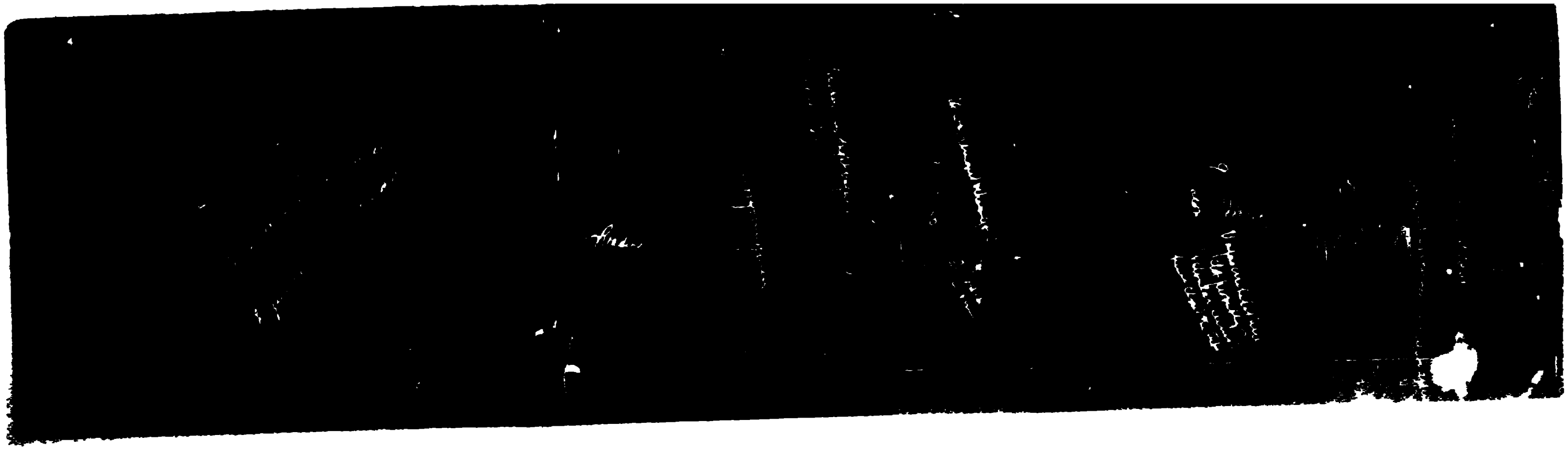


Photo. No. 328. Expt. No. 150. See pages 116 and 243. Ludwig tracing II. Reads from left to right.

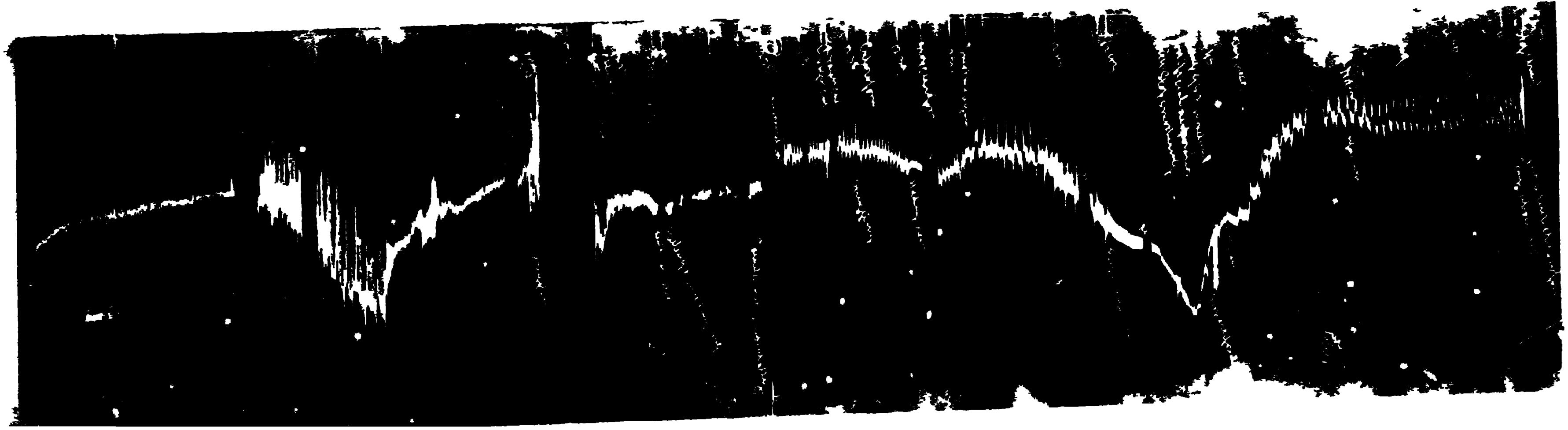


Plate II.

Photo. No. 326. Expt. No. 150. Fick tracing I.
Reads from right to left. See pages 116 and 243.

Photo. No. 334. Expt. No. 151. Fick reading I. Reads from right to left. See page 118.

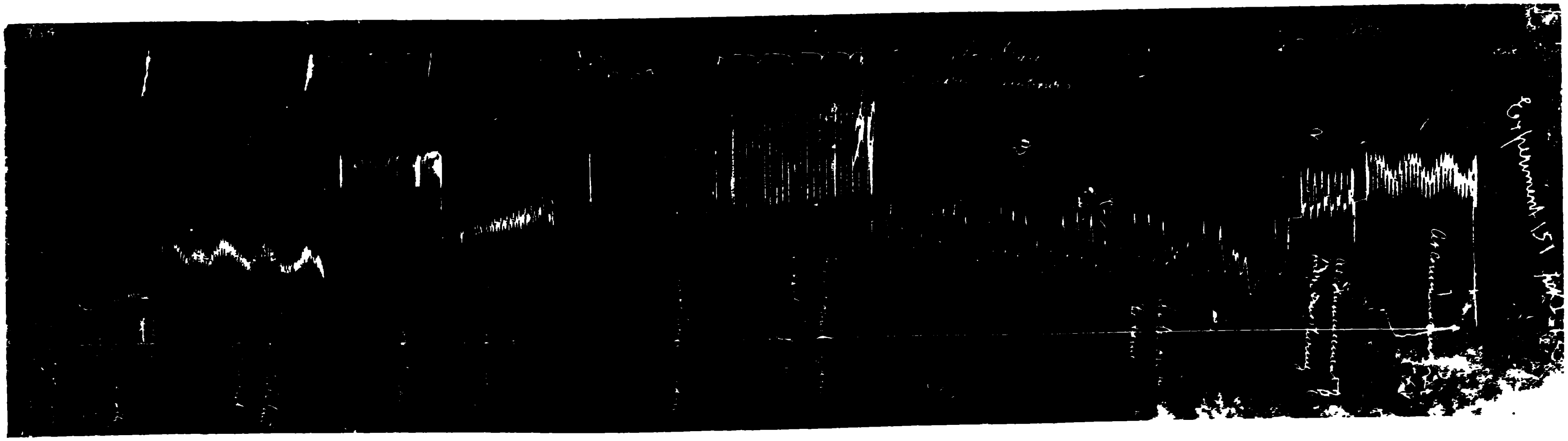


Plate III.

Photo. No. 340. Expt. No. 156. Ludwig tracing I. Reads from left to right. See page 120, especially observation C.

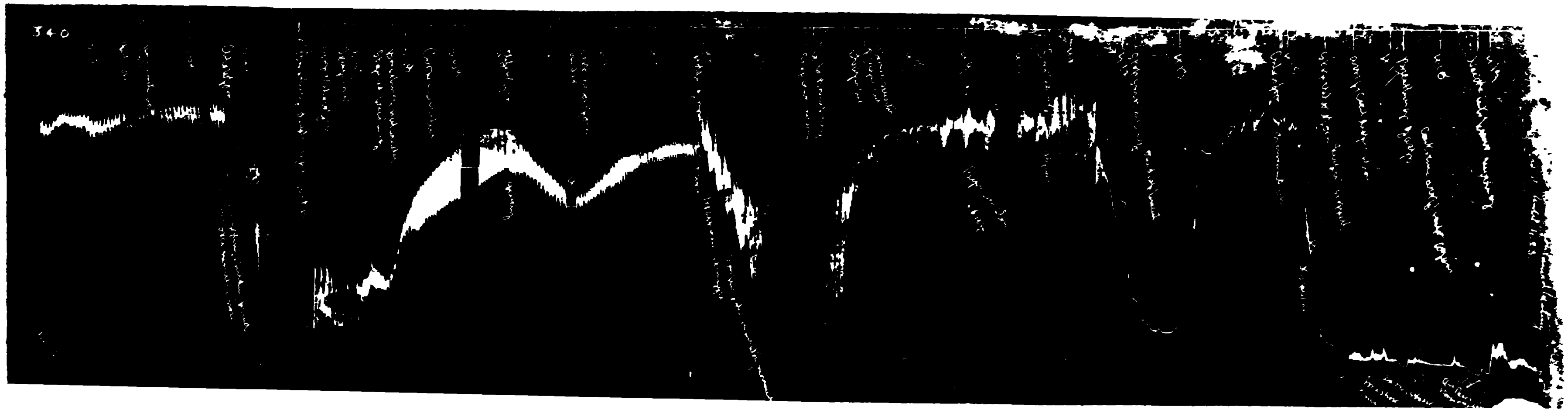


Photo. No. 341. Expt. No. 156. Fick tracing I. Reads from right to left. See observation C, page 120.

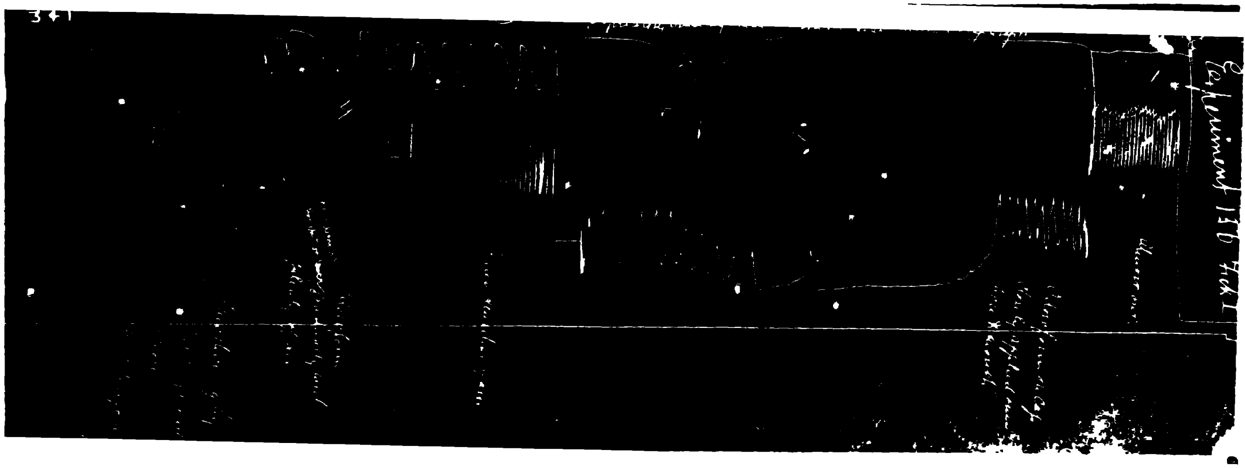


Plate IV.

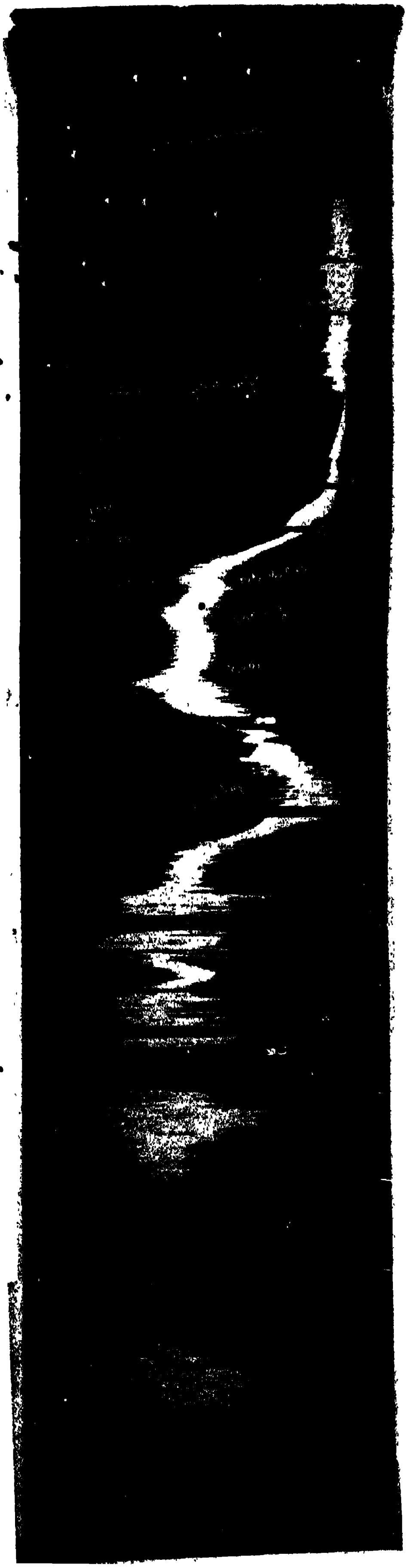


Photo. No. 309. Expt. No. 143. Ludwig tracing I. Reads from left to right. See pages 110 and 393.

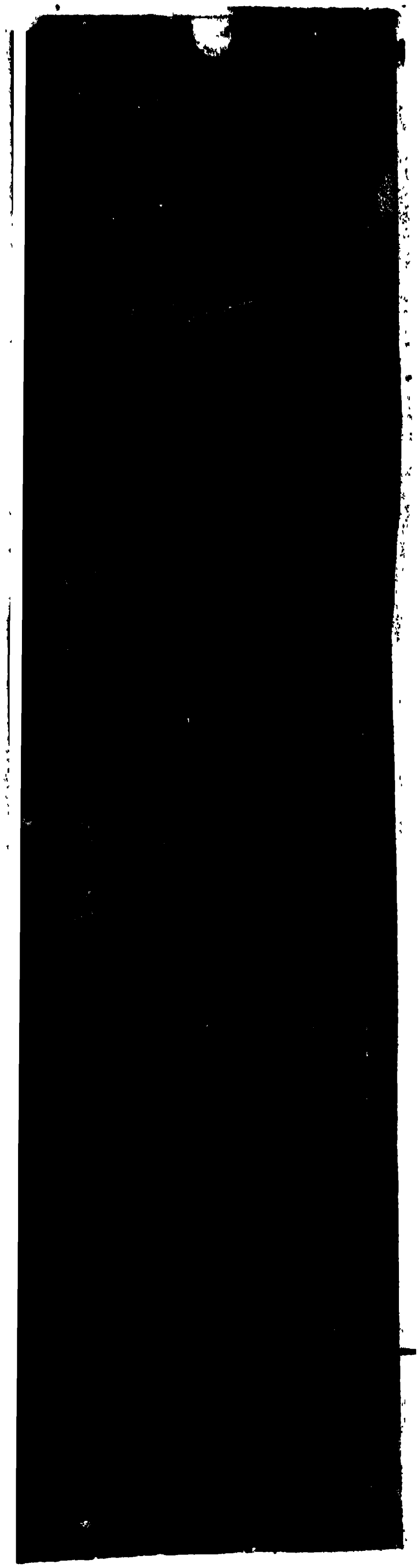


Photo. No. 112. Expt. No. 64. Ludwig tracing III. Reads from left to right. See page 53.

Chloroform and vagus irritation stopped, at this point.

Plate V.

Photo. No. 318. Expt. No. 148. Ludwig tracing I. Reads from left to right. See pages 113 and 342.

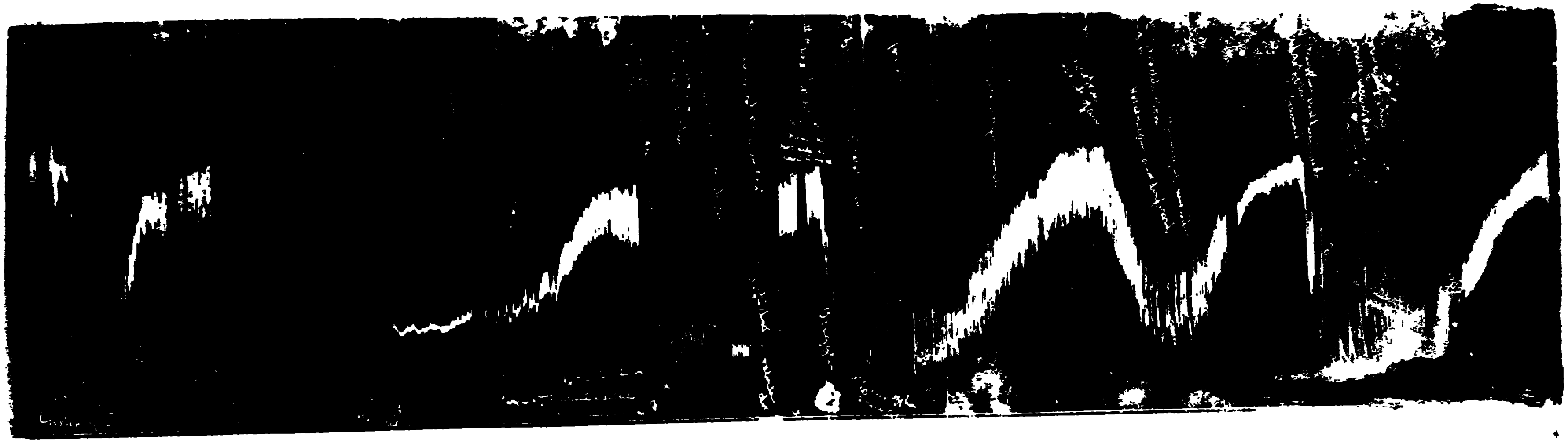


Photo. No. 319. Expt. No. 148. Ludwig tracing II. Reads from left to right. See pages 113 and 342.

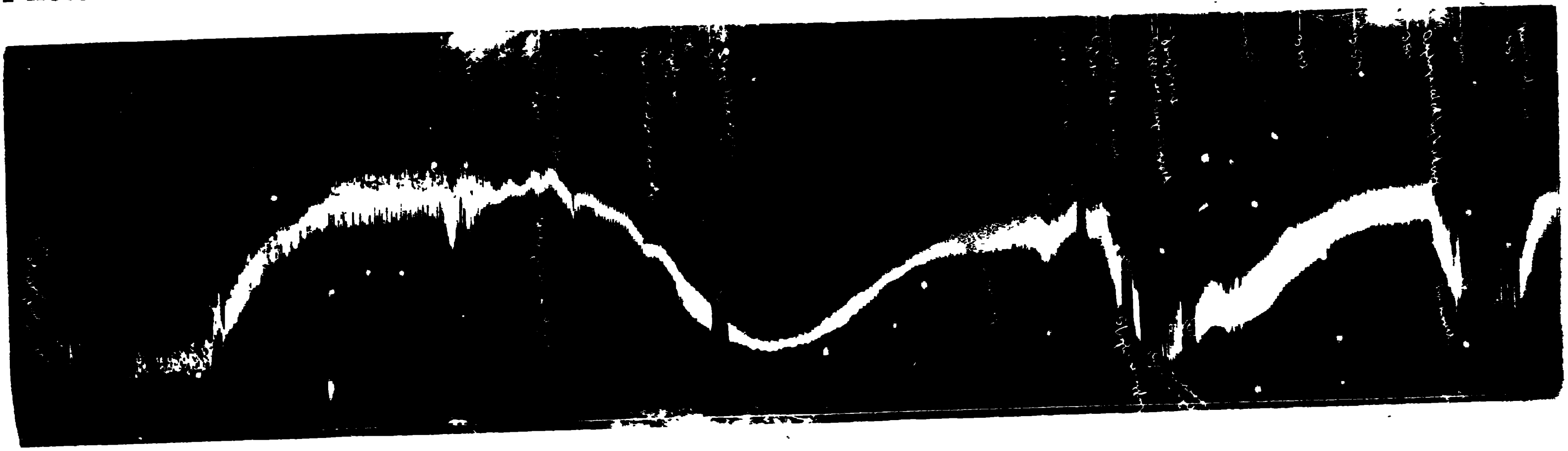


Plate VI.

Photo. No. 322. Expt. No. 148. Fick tracing II. Reads from right to left. See page 113.

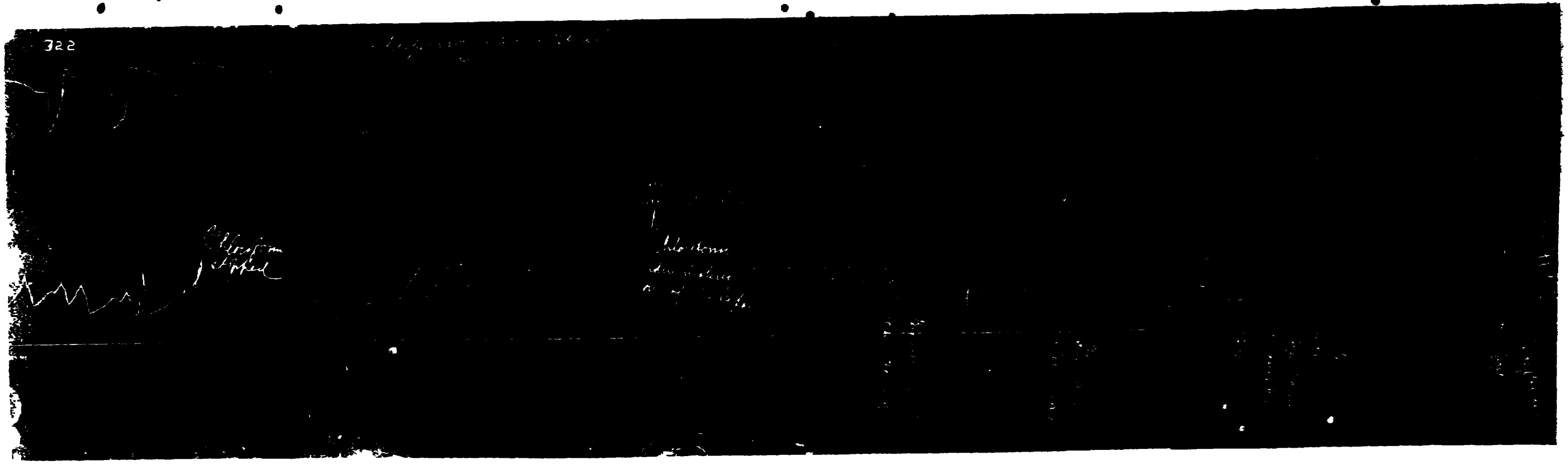


Photo. No. 323. Expt. No. 148. Fick tracing III. Reads from right to left. See page 113.

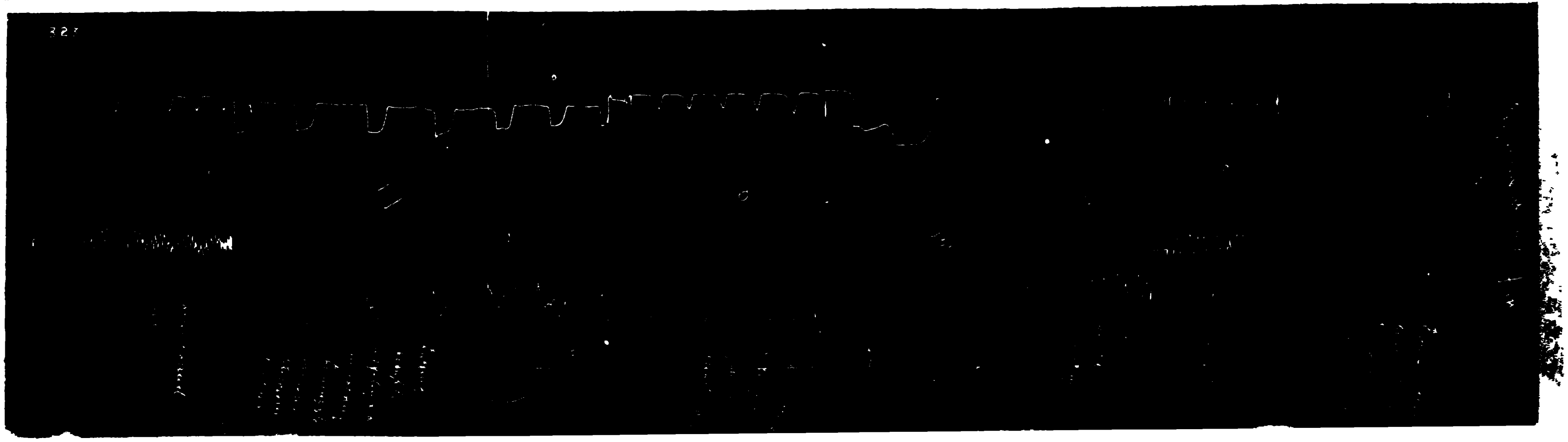


Plate VII.

Photo. No. 384. Expt. No. 178. Ludwig tracing II. Reads from left to right. See page 134.

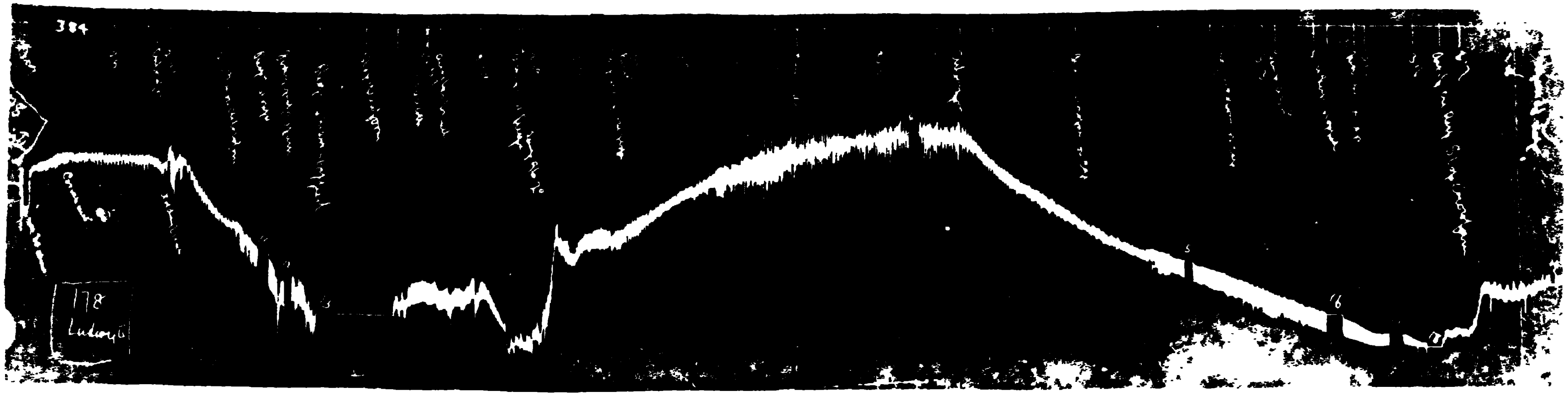


Photo. No. 386. Expt. No. 178. Fick tracing I. Reads from right to left. See pages 120 and 134.

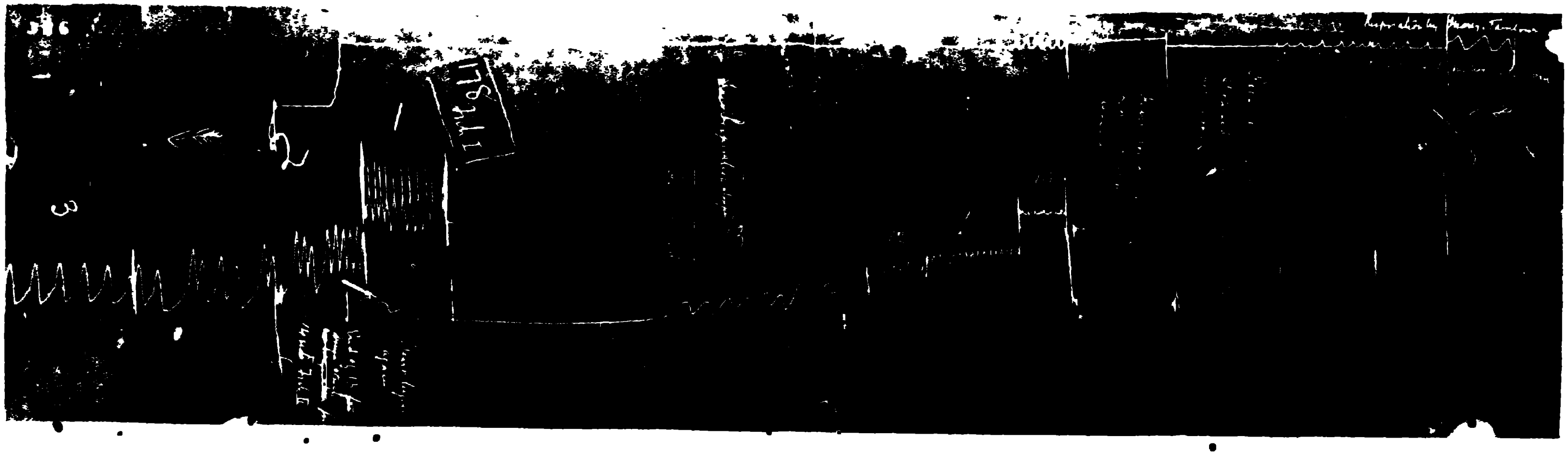


Plate VIII.

Photo. No. 267. Expt. No. 117. Fick tracing III. Reads from right to left. See pages 93 and 120.

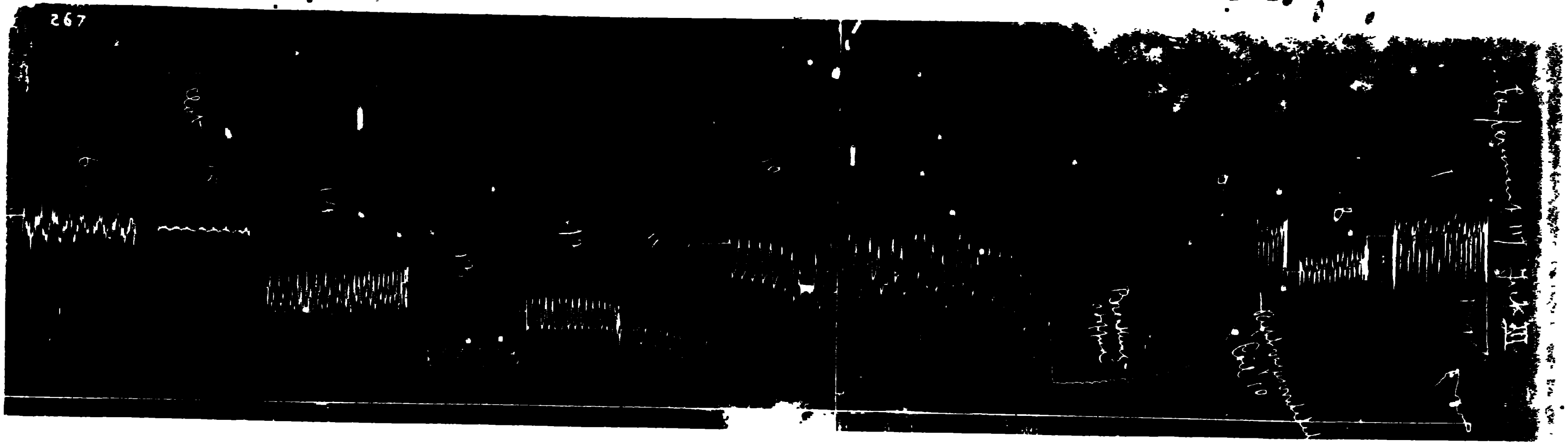


Photo. No. 415. The Glasgow trace. Reads from right to left. See pages 120 and 352.

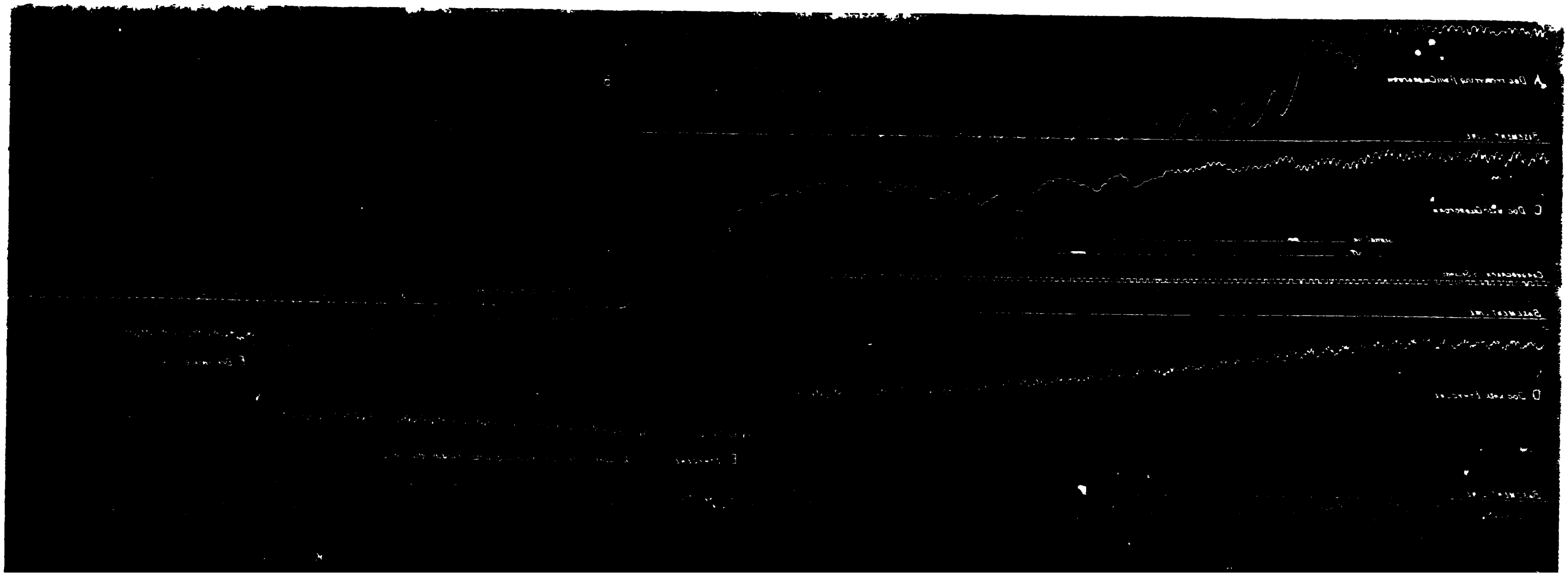


Plate IX.

Photo. No. 421. (Continued on Plate XI.) Expt. No. 186.

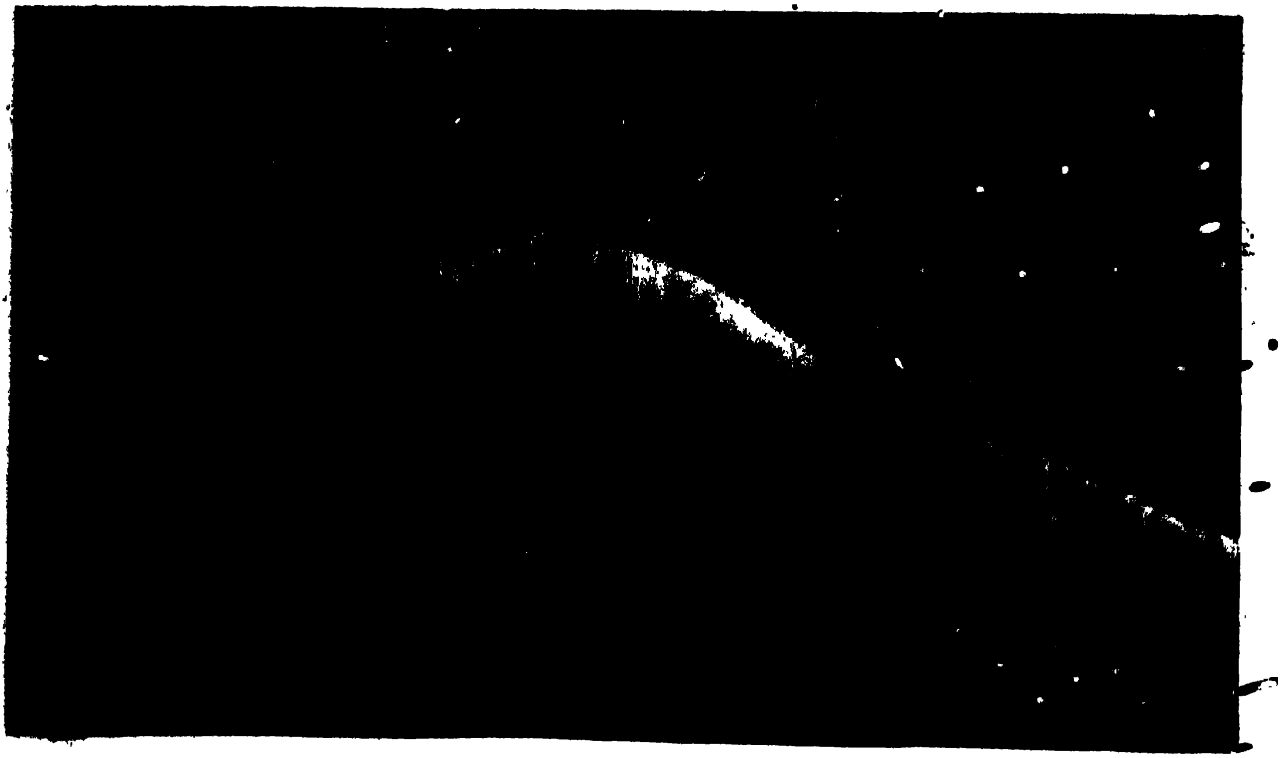


Photo. No. 422. (Continued on Plate XI.) Expt. No. 186.

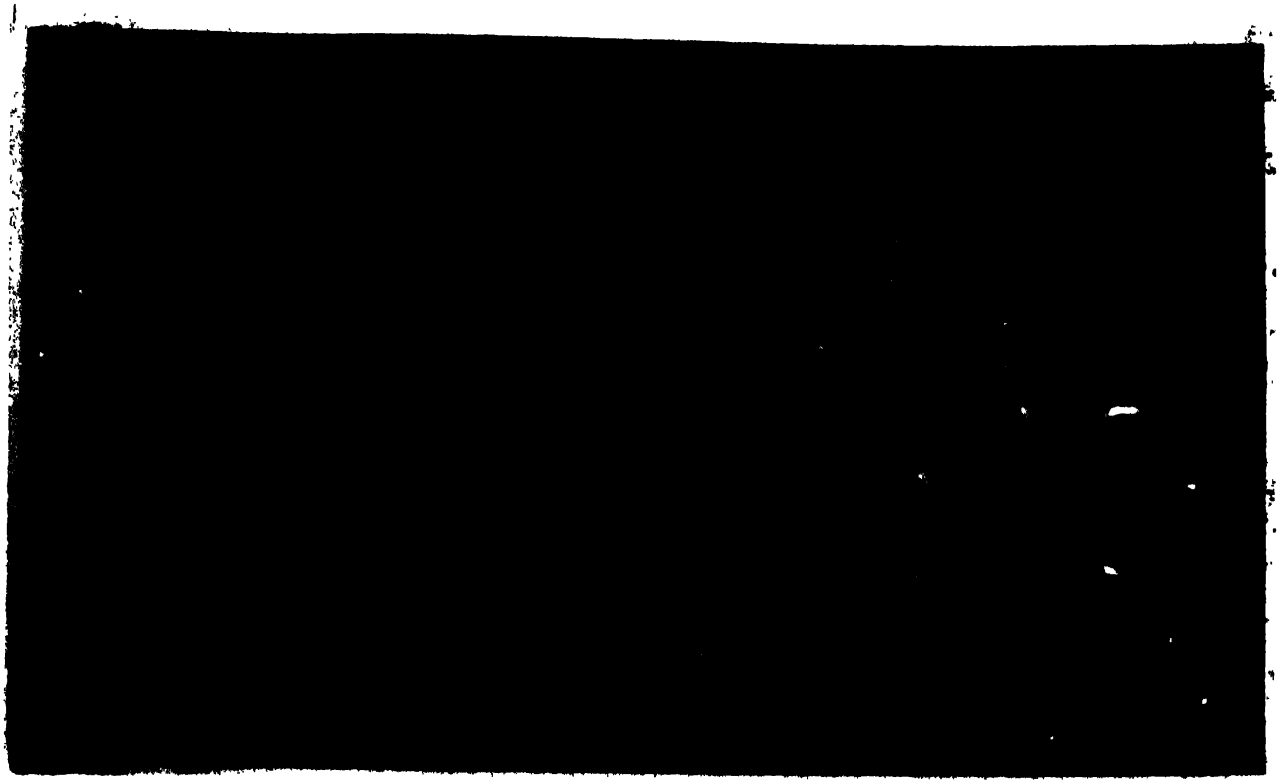
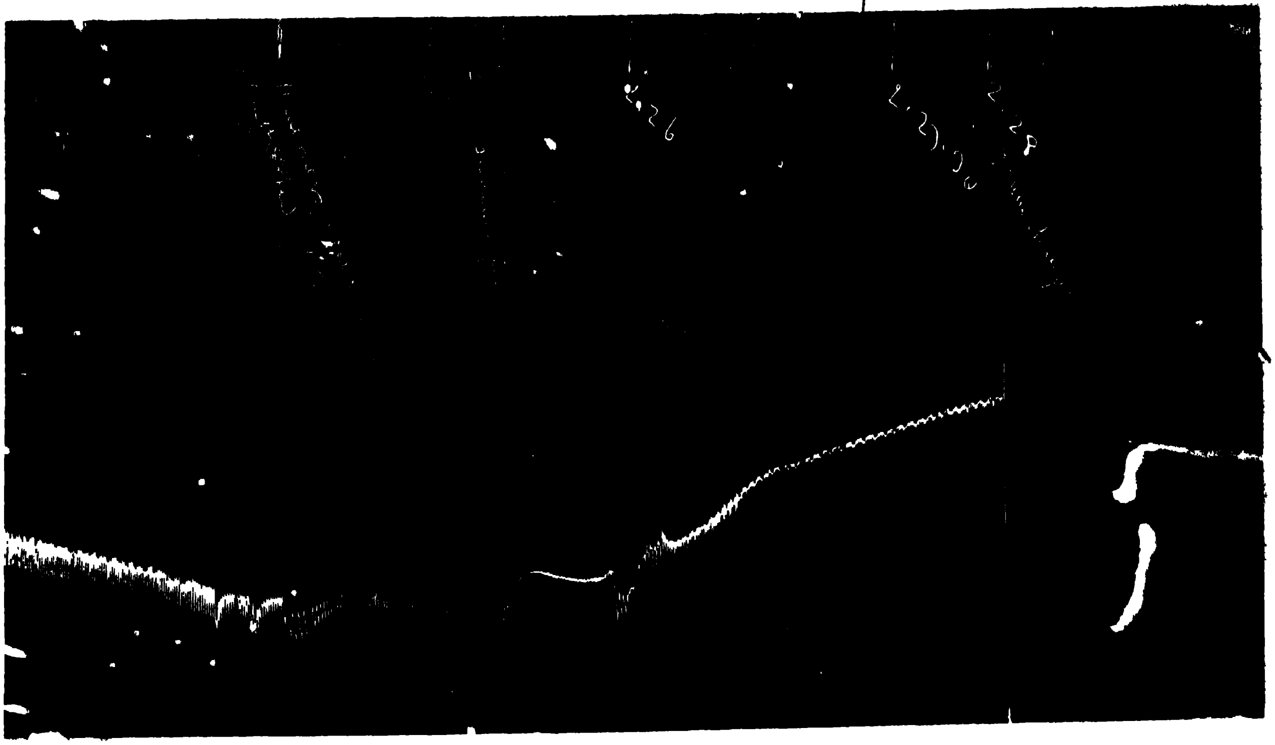


Plate X.

Ludwig tracing I. Reads from left to right. See pages 141, 336 and 354



Ludwig tracing II. Reads from left to right. See pages 141, 336 and 354.

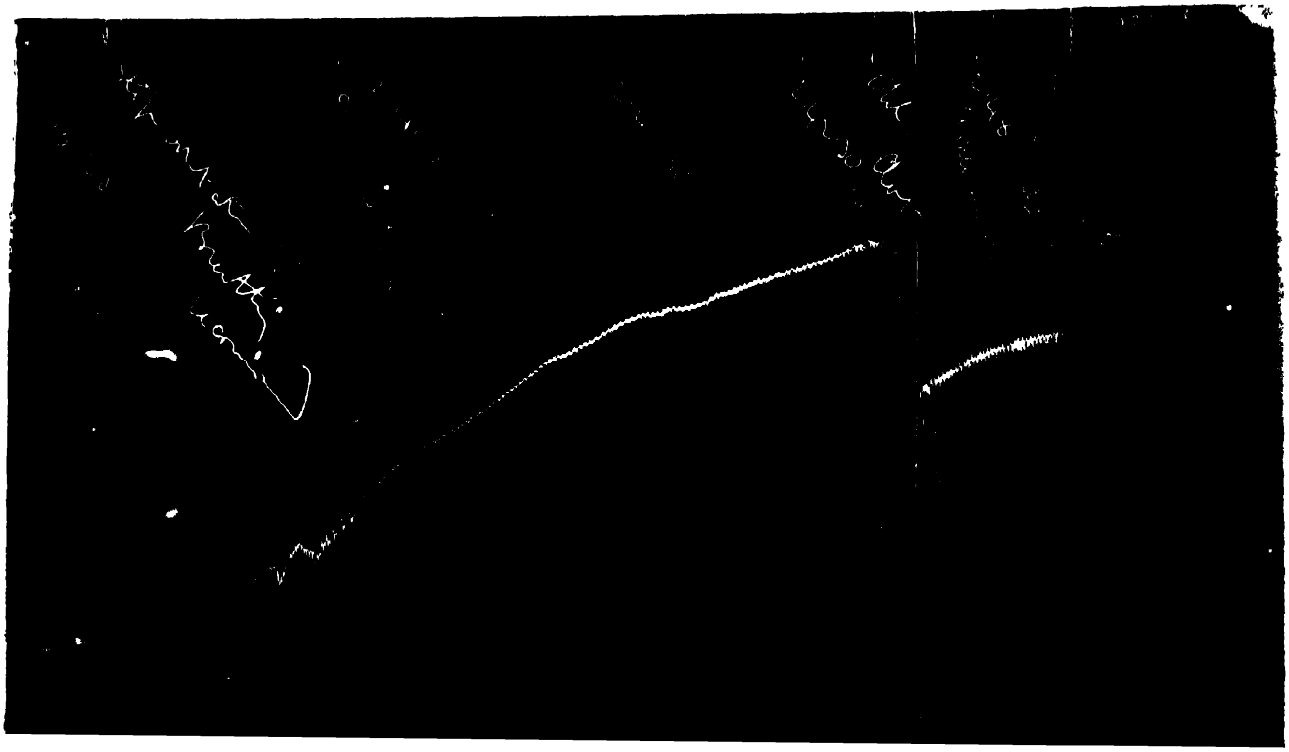


Plate XI.

Photo. No. 423. (Continued on Plate XIII.) Expt. No. 186. Ludwig tracing III.

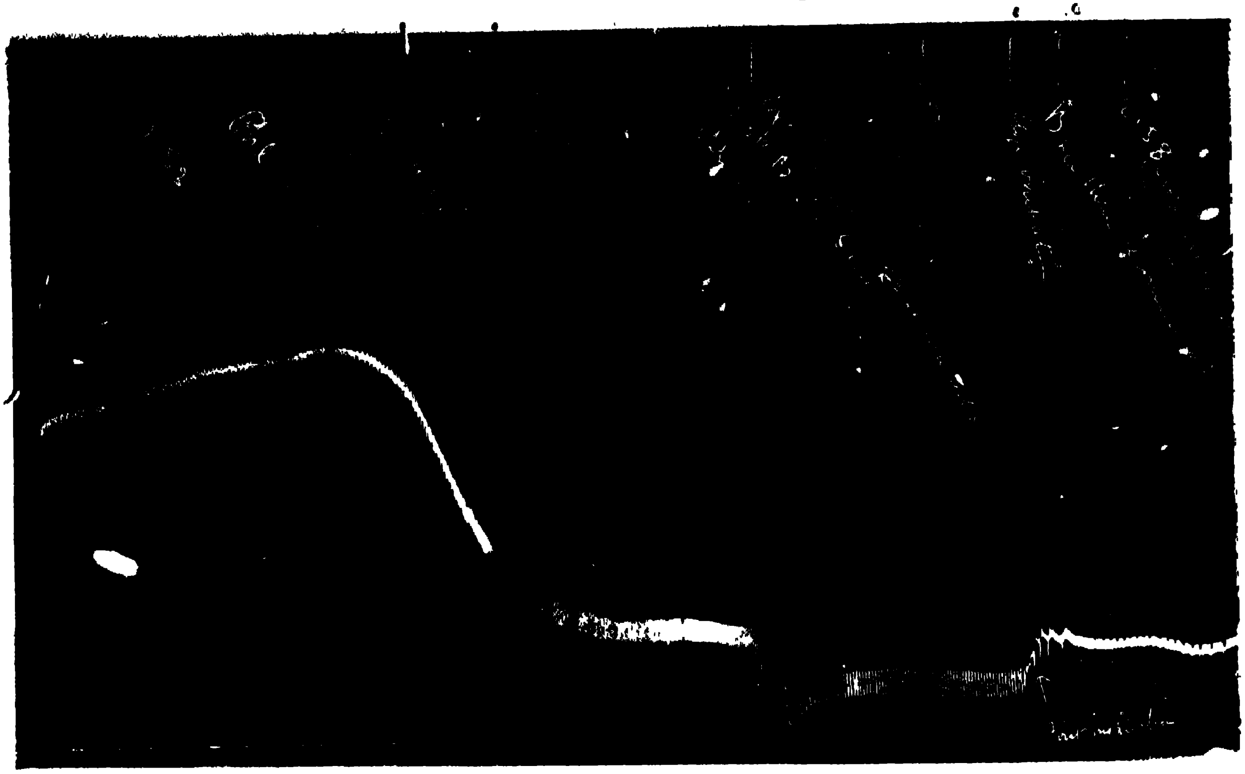


Photo. No. 424. (Continued on Plate XIII.) Expt. No. 186. Ludwig tracing IV.

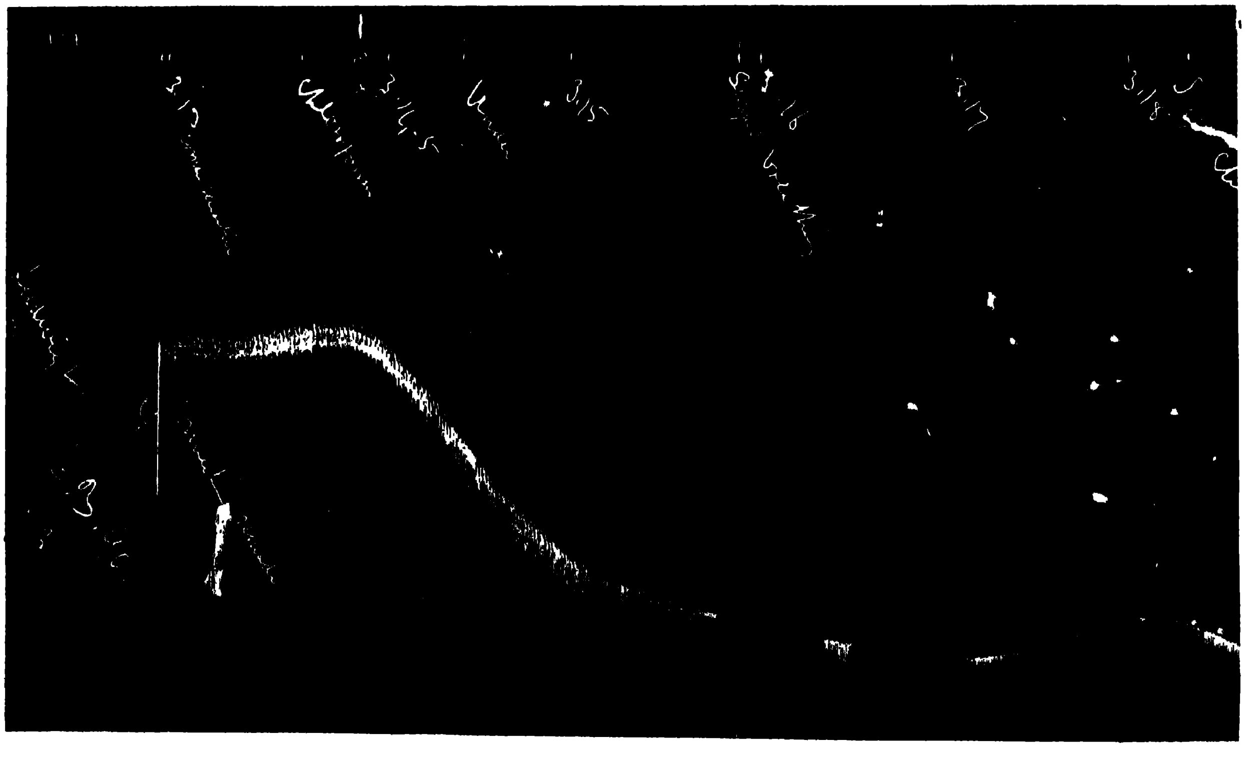
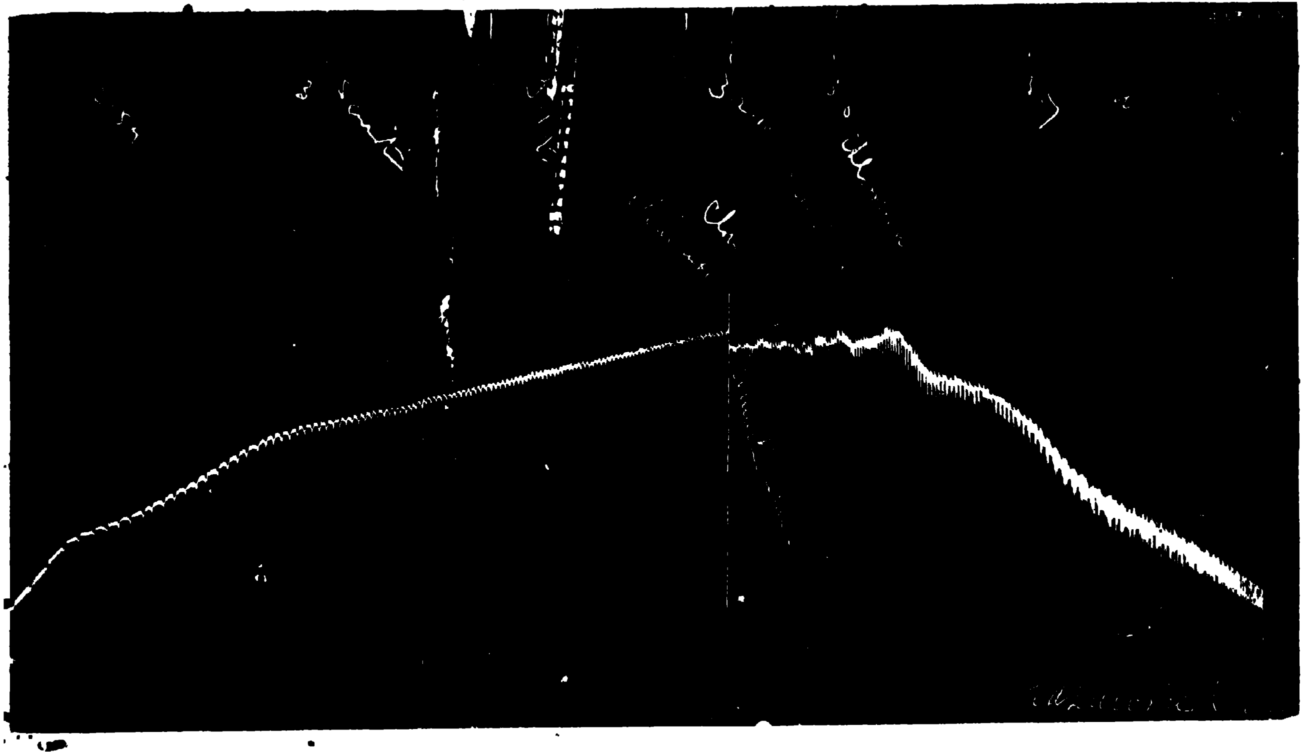


Plate XII.

Reads from left to right. See pages 141, 336 and 354.



Reads from left to right. See pages 141, 336 and 354.

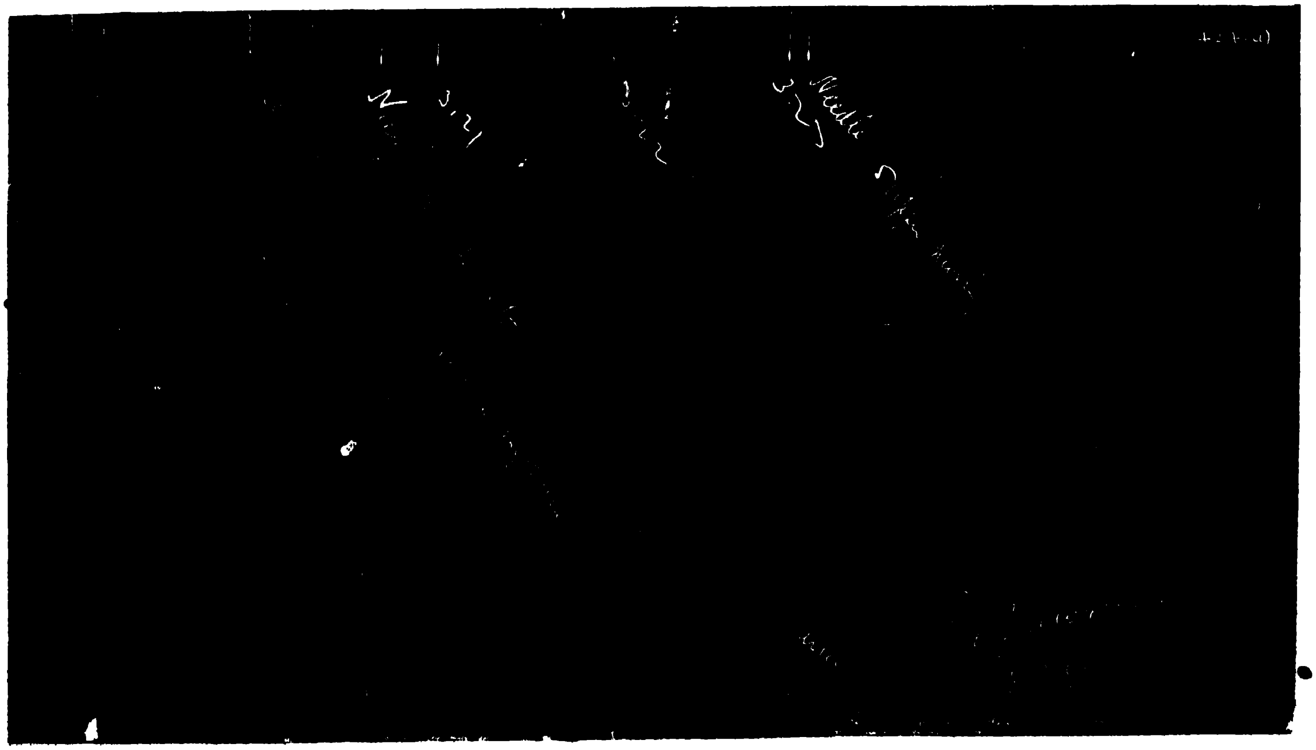


Plate XIII.

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